JOURNAL

OF THE

American Veterinary Medical Association

FORMERLY AMERICAN VETERINARY REVIEW

(Original Official Organ U. S. Vet. Med. Ass'n.)

H. Preston Hoskins, Secretary-Editor, 716 Book Building, Detroit, Mich.

T. A. Sigler, President, Greencastle, Ind. M. Jacob, Treasurer, Knoxville, Tenn.

Executive Board

Geo. Hilton, 1st District; T. E. Munce, 2nd District; D. S. White, 3rd District; J. R. Mohler, 4th District; C. H. Stange, 5th District; Geo. H. Hart, 6th District; Cassius Way, Member at Large, Chairman.

Subcommittee on Journal

D. S. White J. R. Mohler

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Vol. LXXI, N. S. Vol. 24 June, 1927 No. 3

A WONDERFUL EDITORIAL

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We are pleased to take this opportunity to thank the editor of *The American Herdsman* for his editorial. We thank him on behalf of every member of the veterinary profession. We congratulate him upon the keenness of his vision, the clarity of his statements and the reasonableness of his arguments. May we hear more from him.

Mr. I. N. Weaver, father of Dr. D. S. Weaver (K. C. V. C. '16), of Coon Rapids, Iowa, died March 29, 1927, at the age of 78 years.

PERSONALS

MARRIAGE

Dr. Frank M. Wilson (Chi. '11), to Miss Pagie Page, both of Mechanicsville, Iowa, March 26, 1927.

BIRTH

To Dr. and Mrs. D. S. Weaver, of Coon Rapids, Iowa, a daughter, Patricia Ann, March 26, 1927.

PERSONALS

Dr. Robert L. Galt (U. P. '23) has entered practice in Quarryville, Pa.

Dr. C. C. Officer (Colo. '14) has removed from Grapeland, Texas, to Ferris, same state.

Dr. Guy E. Bradham (Ind. '11), formerly of Trenton, Ill., has located at Melvin, Ill.

Dr. W. E. Simonsen (K. S. A. C. '12) has removed from Cherokee, Iowa, to Quimby, Iowa.

Dr. J. H. Patterson (K. C. V. C. '14), formerly of Hillview, Ill., is now at White Hall, Ill.

Dr. Arthur C. Davidson (Corn. '26) has removed from Gouverneur to Edwards, N. Y.

Dr. Charles W. Fisher (Ind. '17) has removed from Martinsville, Ind., to Royal Center, Ind.

Dr. L. O. Fish (Ind. '16), formerly of Norman Station, Ind., removed to Spencer, Ind., April 1.

Dr. E. D. Martin (O. S. U. '11) gives his new address as 36 Third Street, S. E., Carrollton, Ohio.

Dr. O. A. Cook (Chi. '17), formerly of Henry, Ill., is now in Chicago. Address: 1639 East 67th Street.

Dr. S. S. Morgan (K. C. V. C. '12), formerly of Richmond, Missouri, is now practicing in Tampa, Fla.

Dr. Clarence C. Hisel (K. C. V. C. '16), of Enid, Okla., has been appointed state veterinarian of Oklahoma.

Dr. M. M. Fletcher (Chi. '06), of Peoria, Ill., is reported to have sold his practice on account of ill health.

Dr. Roy W. Broadhurst (San Fran. '13), formerly of Long Beach, Calif., is now located at Canutillo, Texas.

Dr. E. A. Buxton (Chi. '90), of Vinton, Iowa, has been re-elected Mayor of his town, carrying every precinct.

Dr. H. A. Meisner (U. P. '90), a practitioner of Baltimore for many years, is now located at Rodger's Forge, Md.

Dr. C. J. Marshall (U. P. '94) spent Easter vacation on his farm at "Towner Hill," Bradford County, Pennsylvania.

Dr. B. E. Grover (Chi. '12) has sold his practice at West Branch, Iowa, and entered practice again at Pleasantville, Iowa.

Dr. Peter Garside (Chi. '10), who has been at Daytona Beach, Florida, during the winter, has returned to Bourbon, Ind.

Dr. E. L. Cornman (U. P. '00), of Marietta, Pa., spent Easter with a classmate, Dr. Grant Catlatt, near Strasburg, Virginia.

Dr. A. L. Brown (Corn. '15), who has been in practice at Walton, N. Y., has accepted a position with the Borden Company.

Dr. N. F. Williams (K. C. V. C. '11) has been reappointed state veterinarian of Texas by the new governor of the Lone Star State.

Dr. T. J. Stover (Ont. '01), formerly of Arcadia, Calif., is now located in Pasadena, Calif. Address: 262 S. Roosevelt Avenue.

Dr. A. E. Harding (Ont. '16), formerly of Jerseyville, Ill., has purchased the practice of Dr. L. V. Puckett, at Mt. Vernon, Ill.

Dr. C. G. Glendinning (Ont. '89), formerly of Clinton, Ill., and Fort Lauderdale, Fla., has entered the real estate business at Decatur, Ill.

Dr. L. V. Puckett, of Mt. Vernon, Ill., has been appointed Adams County (Ill.) Veterinarian, succeeding Dr. Charles W. Gates, resigned.

Dr. Wm. Herbert Lowe (Amer. '88), of Paterson, N. J., was recently elected a member of the Board of Directors of the Paterson Rotary Club.

Dr. J. F. DeVine (Amer. '98), of Goshen, N. Y., judged the hunters and saddle horses at the Pinehurst (N. C.) Horse Show, April 4-15, 1927.

Dr. Arch Freer (Corn. '13), who has been located at Charleston, Miss., for some time, has returned to New York State. He is now located at Jeffersonville.

Dr. I. L. Buchanan (Corn. '09) recently gave a talk on "Jurisprudence" before the senior class of the New York State Veterinary College at Cornell University.

Dr. C. A. Forbes (Chi. '15), of Princeton, Ill., has been employed by Marshall and Putnam counties (Ill.) to conduct tuberculin tests. He is located at Henry, Ill.

Dr. J. R. Taylor (Chi. '04), of Sullivan, Ill., has been employed as Fayette County (Ill.) Veterinarian, at a salary of $3,600 per year. He assumed his duties April 1.

Dr. B. A. Brian (K. C. V. C. '17), who has been located in Belle Plaine, Kansas, for the past ten years, has removed to Minonk, Ill., and entered practice there.

Dr. David A. Benson (Gr. Rap. '18), of Bark River, Mich., has accepted a position with the U. S. Bureau of Animal Industry and been assigned to meat inspection work in Chicago.

Dr. John Mudd (McK. '18), of Stronghurst, Ill., has received the appointment as Cass County (Ill.) Veterinarian. He entered upon his duties April 1, with headquarters at Virginia, Ill.

Dr. S. A. Ridgway (Ind. '03) has accepted the position of Kane County (Ill.) Veterinarian, at a salary of $4,000 per year. Dr. Ridgway was formerly Marion County (Ill.) Veterinarian.

Dr. Joseph W. Vansant (U. P. '02), of Fox Chase, Pa., has issued a call for a reunion of his class, to be held at the University of Pennsylvania Veterinary School on Alumni Day, June 11, 1927.

Dr. E. B. Parker (K. C. V. C. '17), of Carthage, Ill., has resigned his position as Henderson County Veterinarian to accept an appointment in the U. S. Bureau of Animal Industry, at Chicago.

Dr. W. E. LeCroy (Ind. '14), who has been located at Stronghurst, Ill., as Henderson County Veterinarian, has resigned to accept a similar position in Marion County, with headquarters at Salem, Ill.

Dr. N. S. Mayo (Chi. '89), of North Chicago, Ill., delivered an address on "The Training and Care of Army Horses," before the officers of the 65th Cavalry Division, March 31, 1927. A large attendance is reported.

Dr. R. G. Flowers (Ont. '06), of Fort Worth, Texas, acted as the official veterinarian at the Southwestern Exposition and Fat Stock Show, March 5-12, 1927. The Show was a record-breaker and one of the most successful ever held.

Dr. Chas. W. Gates, who has been Adams County (Ill.) Veterinarian since 1923, resigned his position, effective January 31, 1927, to accept an appointment in the Bureau of Animal Industry. He will be stationed at Des Moines, Iowa.

Dr. J. S. Anderson (Chi. '94), formerly of Lincoln, Nebr., is now back in private practice in Hastings, Nebr. Dr. Anderson has purchased a residence there and now has under construction a modern hospital, to be fully equipped for the care and treatment of both large and small animals.

Dr. Stanley Brewer (Ind. '18), of Pleasantville, Ind., had a narrow escape from death when his coupe was struck by a train at a road crossing recently. He suffered several cuts and bruises, but was able to walk to the office of a physician for medical attention. The coupe was a total wreck.

Dr. F. L. Harrison (Ont. '07), who has been engaged in general practice at Fairgrove, Michigan, for a number of years, has accepted a position with the Michigan State Department of Agriculture. Dr. Harrison entered upon his new duties April 1, and has been assigned to tuberculin testing and allied work.

Dr. L. C. Drum (Chi. '10), of Gridley, Ill., has been employed as Mountrie County (Ill.) Veterinarian, succeeding Dr. J. R. Taylor, who held the position ever since the organization of county tuberculosis eradication work several years ago. Dr. Drum will receive a salary of $3,000 per year and pay his own expenses.

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STUDENT CHAPTERS

At the meeting in Des Moines, in 1924, the proposal for junior membership in the A. V. M. A. was advanced by Dr. C. H. Stange. The necessary changes in the Constitution and By-laws were given official approval at the meeting in Portland, in 1925. Rules and regulations for student chapters composed of junior members were presented to and approved by the Association at the meeting in Lexington, in 1926, thereby paving the way for the organization of such chapters.

Within the past six months applications have been received, from student organizations in five of our veterinary colleges, for the organization of student chapters of the A. V. M. A. in these institutions. Four of these applications have already been approved by the Executive Board and three of the chapters are already organized and are functioning. The others probably will be before the close of the present college year.

Perhaps the greatest advantage to be gained from the student chapters lies in the opportunity afforded for acquainting veterinarians in the making with the national organization, its history and its traditions, its purpose and its functions. It is to be hoped that in the great majority of cases the young veterinarian will want to become an active member of the Association just as soon as he has terminated his period of junior membership. It would appear to be much easier and certainly much more desirable to have these additions to the veterinary profession continue to have some connection with the national organization rather than to be turned loose, just as soon as graduation is over and perhaps be lost to the A. V. M. A. forever.

It is not unusual for a veterinarian to signify his desire to become a member of the A. V. M. A. ten, twenty, or even thirty years after he has left college. Almost invariably the man will acknowledge that he wishes that he had done so many years before. Some of our members take a great deal of pride in referring to the length of their membership in the Association. There now are three members who can boast a continuous membership of more than fifty years; eleven more who have been members forty years or more, while the number who have been on the roll for a quarter of a century or more is in the neighborhood of 157 at the present time.

We welcome our new student chapters with their several hundred junior members and we look forward to the time when

there will be a chapter in every recognized veterinary college in the United States and Canada. The possibilities for the usefulness of these chapters is almost unlimited.

EXECUTIVE BOARD ELECTION

For the second time in four years, a contingency has arisen in connection with an Executive Board election that is not specifically covered by the By-laws. Section 1 of Article 10 provides that the names of the five members receiving the highest number of nominating votes shall be placed upon the ballot. When the nominating ballots for the election now in progress in District No. 1 (Canada) were counted, the tellers found that there was a three-cornered tie for fifth place. This will explain why our Canadian members found seven names on the ballot instead of the customary five. The nominating polls closed May 16 and the election ballots were placed in the mail the same day. The polls will remain open until July 16, 1927. Brief biographical sketches of the nominees follow:

BAKER, M. C. — Montreal, Quebec

Practitioner. Graduate of Montreal Veterinary College, 1879 and McGill University, 1890. Joined A. V. M. A., 1902. Third vice-president, 1922-23.

BRUCE, E. A. — Agassiz, British Columbia

Animal Pathologist, Health of Animals Branch, Canadian Department of Agriculture. Graduate of Ontario Veterinary College, 1905. Joined A. V. M. A., 1916.

HARGRAVE, J. C. — Medicine Hat, Alberta

Chief Inspector for Alberta, Health of Animals Branch, Canadian Department of Agriculture. Graduate of McGill University, 1895. Joined A. V. M. A., 1902. Resident secretary for Alberta, 1921-27.

HILTON, GEORGE — Ottawa, Ontario

Veterinary Director General of Canada. Graduate of Ontario Veterinary College, 1897. Joined A. V. M. A., 1911. Member of Executive Board, District No. 1, 1918-27; resident secretary for Ontario, 1915-19.

MCGILVRAY, C. D. — Guelph, Ontario

Principal Ontario Veterinary College. Graduate of Ontario Veterinary College, 1900, and McKillip Veterinary College, 1901. Joined A. V. M. A., 1906. Fifth vice-president, 1910-11; fourth vice-president, 1915-16; first vice-president, 1924-25; member of Committee on Glanders, 1913-15; member of Audit Committee, 1917-18; member of Committee on Resolutions, 1918-21; member of Committee on History, 1923-25; member of Committee on Necrology, 1926-27.

MOORE, A. E. — Montreal, Quebec

Chief Travelling Inspector, Health of Animals Branch, Canadian Department of Agriculture. Graduate of McGill University, 1894. Joined A. V. M. A., 1903.

WATSON, E. A. — Ottawa, Ontario

Chief Pathologist, Health of Animals Branch, Canadian Department of Agriculture. Graduate of Ontario Veterinary College, 1904. Joined A. V. M. A., 1911. Second vice-president, 1919-20 and 1925-26; resident secretary for Alberta, 1913-15; member of Committee on Tuberculosis, 1925-27.

APPLICATIONS FOR MEMBERSHIP

See April, 1927, JOURNAL

FIRST LISTING

COPLAND, ALEX. L. Grosse Pointe Farms, Mich.
D. V. M., Michigan State College, 1924
Vouchers: B. J. Killham and H. Preston Hoskins.

CUNNINGHAM, JAMES JOSEPH 1537 Overington St., Frankford, Philadelphia, Pa
V. M. D., University of Pennsylvania, 1925
Vouchers: J. Wm. Linberg, and G. A. Dick.

FINCK, VICTOR HARRY Elizabeth City, N. C.
D. V. M., Chicago Veterinary College, 1916
Vouchers: L. J. Faulhaber and Wm. Moore.

GRAVES, HOBSON WILLIAM Box 341, Union, S. C.
D. V. M., Alabama Polytechnic Institute, 1924
Vouchers: W. K. Lewis and W. A. Barnette.

HAMILTON, CECIL MORLEY 2045 Third Ave., Detroit, Mich.
B. V. Sc., Ontario Veterinary College, 1926
Vouchers: E. E. Patterson and H. Preston Hoskins.

HOGG, JAMES S. 161 Pine St., Oxford, Pa.
V. M. D., University of Pennsylvania, 1924
Vouchers: E. W. Hogg and Thos. D. James.

MACDONALD, HAROLD STEWART CLAUDE 2045 Third Ave., Detroit, Mich.
B. V. Sc., Ontario Veterinary College, 1924
Vouchers: E. E. Patterson and J. E. Patterson.

MCLELLAN, DAVID JOHN 51 Enterprise St., Moncton, N. B.
V. S., Ontario Veterinary College, 1917
Vouchers: A. T. McLean and George Hilton.

PHILLIPS, E. S., Ingalls, N. C.
D. V. M., Kansas City Veterinary College, 1916
Vouchers: A. A. Husman and Wm. Moore.

ROSENBERGER, JOHN NEWTON 749 S. 51st St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1910
Vouchers: T. E. Munce and Henry W. Turner.

TURRELL, CHARLES WILLARD Arabi, La.
D. V. M., Indiana Veterinary College, 1918
Vouchers: G. T. Cole and S. C. Cook.

WATSON, HAROLD L. 159 Main St., Montpelier, Vt.
D. V. M., Grand Rapids Veterinary College, 1915
Vouchers: L. H. Adams and F. M. McConnell.

Applications Pending

SECOND LISTING

Brueckner, Arthur Louis, 5643 Belmar Terrace, Philadelphia, Pa.
Davis, Oscar Gold, Roxboro, N. C.
Dawson, Bert L., 320 Agricultural Bldg., Raleigh, N. C.
Dillahunt, Peter A., Springfield, Ohio.
Everett, Ed., Box 384, Raleigh, N. C.
Hall, Walter J., 642 C St. N. E., Washington, D. C.
Heim, Thomas Brumbaugh, 81 E. College St., Alliance, Ohio.
Junod, Fred L., N. Court St., Athens, Ohio.
Lewis, Irvin T., Box 1305, Charlotte, N. C.
Longacre, William S., 208 N. 3rd St., Lehighton, Pa.
Michels, Charles B., 18a N. 31st St., Belleville, Ill.
Mitcham, Rease, 840 S. Washington Ave., El Dorado, Ark.
Nathan, Simeon Aaron, Chapel Hill, N. C.

Nickel, William Calvin, 166 S. Main St., Nazareth, Pa.
Reed, Jacob O., 812 Washington St., Allentown, Pa.
Shelly, John J., 1316 Easton Ave., Bethlehem, Pa.

REINSTATED

Parrish, Guy M., 320 Agricultural Bldg., Raleigh, N. C.

The amount that shall accompany an application filed this month is $7.91, which covers membership fee and dues to January 1, 1928, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Chicago Veterinary Society. Great Northern Hotel, Chicago, Ill. June 13, 1927. Dr. J. B. Jaffray, Secretary, 2956 Washington Blvd., Chicago, Ill.

California State Veterinary Medical Association. Sacramento, Calif. June 13-14-15, 1927. Dr. E. H. Barger, Secretary, University Farm, Davis, Calif.

Texas A. & M. College Short Course for Veterinarians and State Veterinary Medical Association of Texas. College Station, Texas. June 13-14-15-16-17-18, 1927. Dr. D. Pearce, Secretary, Leonard, Texas.

Kansas City Association of Veterinarians. New Baltimore Hotel, Kansas City, Mo., June 20, 1927. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.

Eastern States Tuberculosis Eradication Conference. Hotel Chelsea, Atlantic City, N. J. June 20-21-22, 1927.

New Jersey, Veterinary Medical Association of. Hotel Chelsea, Atlantic City, N. J. June 20-21-22, 1927. Dr. G. P. Ellice, Secretary, Rutherford, N. J.

Keystone Veterinary Medical Association. Philadelphia, Pa. June 22, 1927. Dr. C. S. Rockwell, Secretary, 5128 Chestnut St., Philadelphia, Pa.

Nevada State Veterinary Association. Reno, Nev. June 22-23-24, 1927. Dr. Edward Records, Secretary, University of Nevada, Reno, Nev.

American Association for the Advancement of Science, Pacific Division. Reno, Nev. June 22-23-24, 1927.

Northeastern Pennsylvania Veterinary Medical Club. Montrose, Pa. June 25, 1927. Dr. Thos. D. James, Secretary, 816 N. Main Ave., Scranton, Pa.

Washington, Oregon and British Columbia Veterinary Medical Associations. Bellingham, Wash. June 27-28-29, 1927. Dr. J. W. Kalkus, Secretary, Puyallup, Wash.

Lake States Tuberculosis Eradication Conference. Olds Hotel, Lansing, Mich. June 27-28, 1927.

Michigan State Veterinary Medical Association. Michigan State College, East Lansing, Mich. June 28-29, 1927. Dr. E. K. Sales, Secretary, 535 Forest St., East Lansing, Mich.

Vermont Veterinary Medical Association. Wells River, Vt. June 28-29, 1927. Dr. G. N. Welch, Secretary, Northfield, Vt.

New York State Veterinary Medical Society. Watertown, N. Y. June 28-29-30, 1927. Dr. C. E. Hayden, Secretary, 110 Irving Place, Ithaca, N. Y.

North Carolina State Veterinary Medical Association. Durham, N. C. June 29-30, 1927. Dr. W. T. Scarborough, Secretary, 320 S. Blount St., Raleigh, N. C.

Missouri Valley Veterinary Association. Elks Club, Omaha, Nebr. July 11-13, 1927. Dr. E. R. Steel, Secretary, 8102 Walnut St., Kansas City, Mo.

Maine Veterinary Medical Association. Augusta, Me. July 13, 1927. Dr. C. F. French, Secretary, Rockland, Me.

North Dakota Veterinary Medical Association. Fargo, N. D. July 13-14, 1927. Dr. H. L. Foust, Secretary, State College, Fargo, N. D.

Virginia State Veterinary Medical Association. Ocean View, Va. July 14-15, 1927. Dr. W. H. Ellett, Secretary, Midlothian, Va.

Minnesota State Veterinary Medical Assoiation. University Farm, St. Paul, Minn. July 14-15, 1927. Dr. C. P. Fitch, Secretary, University Farm, St. Paul, Minn.

South Carolina Association of Veterinarians. Spartanburg, S. C. July 19-20, 1927. Dr. M. R. Blackstock, Secretary, 157 Hampton Ave., Spartanburg, S. C.

Illinois State Veterinary Medical Association. Bloomington, Ill. July 20-21, 1927. Dr. W. H. Welch, Secretary, Lexington, Ill.

Missouri Veterinary Medical Association. Fulton, Mo. July 27-28-29, 1927. Dr. J. D. Ray, Secretary, 400 New Centre Bldg., Kansas City, Mo.

Montana Veterinary Medical Association. Helena, Mont. July 28-29, 1927. Dr. Hadleigh Marsh, Secretary, Livestock Sanitary Board, Helena, Mont.

American Veterinary Medical Association. Bellevue-Stratford Hotel, Philadelphia, Pa. Sept. 13-14-15-16, 1927. Dr. H. Preston Hoskins, Secretary, 716 Book Bldg., Detroit, Mich.

INFECTIOUS CANINE MENINGO-ENCEPHALITIS (FRIGHTS DISEASE)*

By J. C. WRIGHT, *Atlanta, Ga.*

Frights disease or running fits is an acute, infectious and, we think, contagious disease of the central nervous system. It is characterized by deranged consciousness, disturbances of the intestinal tract and, in many cases, disturbances of other functional organs. In so far as we have been able to determine, the disease appeared first in the southern states. Its occurrence is widespread over the southern states and is rapidly spreading northward.

HISTORY

I first noticed this disease in this locality six years ago, in a prison camp, northwest of Atlanta, Georgia, about fifteen miles away. The first subjects infected were a pack of bloodhounds. One of the prison guards shot one of these dogs, thinking it was rabid. The other two dogs ran away or they would have been killed at the same time. After the fits subsided, they returned to the camp, apparently normal. These dogs were isolated on their return to camp and the owner came to me for information concerning them, giving me the symptoms. From the symptoms given, I diagnosed the dogs as not being rabid. I instructed the owner to bring the dogs to my hospital. Upon examination, I found the dogs apparently normal. The dogs were isolated, during which time they had a number of fits.

The symptoms were treated successfully and the dogs were returned home in ten days cured and, to my knowledge, had no recurrence of the disease. From this time on, the number of cases has increased rapidly, both in this and other localities. Today it is prevalent in all the southern states and in many northern, eastern and western states. Many good dogs were killed, the owners thinking them rabid. Each year, the virulence of the disease has become stronger until now it is fatal in approximately 25 per cent of the cases, where the dogs do not reach the semi-comatose or comatose stage or the cases do not become complicated with distemper, pneumonia or hemorrhagic enteritis. Seventy-five per cent are fatal where complications of hemorrhagic enteritis or distemper appear. Practically 100 per cent

*Presented at the sixty-third annual meeting of the American Veterinary Medical Association, Lexington, Kentucky, August 17-20, 1926.

are fatal where the dogs enter the semi-comatose or comatose state.

Symptoms

A languid condition is one of the first symptoms noticed. In some cases, one of the first symptoms noticed is a deranged appetite. Clinical symptoms are as follows: Temperature, 102.5 to 104.5° F. Respiration altered, depending upon the duration of the disease. Pulse very full in the first stages, quickened and, in some cases, intermittent. In the last stages of the disease the pulse is generally small and intermittent. The membrana nictitans has a bluish tinge. The conjunctivae are generally injected. The membranes of the mouth and tongue are pale at the outset, turning to a bluish tinge in the last stages. The hair-coat is generally rough and the skin dry. The eyes have a starey, glassy look and after the animal has a fit, his face betrays anxiousness.

As the disease progresses, the eyes recede slightly, due to the wasting away of the orbital pad and facial muscles. At this stage, the eyes generally become catarrhal. At the onset of the disease, the dog has a variable appetite, often eating voraciously, other times refusing food. After the dog is once down, his appetite is completely gone. Animals in this state can not in many cases retain nourishment by forced feeding. In the milder form of fits, our attention is called to the following symptoms: For no apparent reason, the dog becomes frightened, starts running around, clockwise and counter-clockwise, if in close quarters. If at large, the dog runs off and seeks dark, out-of-the-way places to hide; others exhaust themselves by running, barking, yelping and howling. Amaurosis is apparent as the animals run into, over and against objects. In the milder form, you get from one to twenty of these spells per day. They may skip several days, decreasing in severity as the dogs tend toward recovery. If the animal turns for the worse, the fits increase, both in number and in severity, until the semi-comatose stage is reached. After the animal reaches this stage, he has convulsions almost continuously, growing weaker after each convulsion and often yelping continuously.

In lapsing into a convulsion, the dog's eyes roll back beneath the upper lids and the labial muscles will be tensely drawn, showing the teeth as though snarling. The head is generally drawn backwards. The animal champs his jaws frequently,

frothy saliva exudes from between his lips; his legs work as if running; involuntary urination and defecation often take place during this time. After the dog is relaxed from the convulsion, he often attempts to get up and run but falls exhausted, breathing fast and deep. The pulse at this time is irregular, often running as high as 250 per minute. During this stage, the dog rapidly becomes emaciated. Then convulsions often take place in rapid succession and are ushered in by noises, such as rattling of pans, loud speaking or talking or touching the animal. After the dog reaches this stage, he does not live over three to five days. In this stage, diarrhea is very prominent, the fecal matter is fetid, gaseous and mixed with blood. We might add that when the dog does not go into the semi-comatose or comatose stage but continues to run and bark with these fits, each spell seems to become lighter in severity and the dog generally recovers in from ten days to three weeks.

We spoke of amaurosis being present in the dogs. This condidition lasts only during and directly following the first stage, for only a short period of time. A collie pup entered my hospital with a history of frights disease. The dog was blind at the time he entered the hospital. The owner was asked if the pup had always been blind. She replied that the pup went blind shortly after he took this disease. On examination of the eye, no external symptoms were seen. The cornea was normal; the lens did not show any malformation or blemishes; the fluid in the anterior chamber appeared normal; the retinae also appeared normal. The pupils were dilated and no atropin had to be used in an examination of the deeper structure of the eyes. The pup's expression was blank. He found his way around by smell and by feel. He continued to have fits until the owner killed him. On autopsy, the typical symptoms were found. On examining the optic structures, it was found that the optic nerves were atrophied just anterior to where they come off the optic chiasm.

Other symptoms of the disease that we noted were purulent dermatitis and ulcerated keratitis. The purulent dermatitis is caused by *Staphylococcus aureus*. We did not try to get a culture from the ulcerated eyes. Two cases of purulent dermatitis were noted. One animal died but the other made a complete recovery. In the cases where ulcerated keratitis followed the catarrhal inflammation of the eyes and where the purulent dermatitis was at its height, the eyes ulcerated. In each case the cornea was eaten through except for Descemet's membrane, which

protruded, causing staphyloma. These were operated on without success as the fluid in the anterior chamber of the eye was affected. In the period following the convalescent stage, we have had occasion to note secondary anemia in nine cases. In these cases the animals were very weak, mucous membranes very pale, and the animals' appetites very poor for the most part. In two cases, the dogs were completely down with the disease. Under treatment, they responded after a long period of time.

One peculiar case which was singular in type came under our observation. The dog entered our hospital with fits and was recovering fast when a relapse set in. The animal's temperature would go up to 105.6° F. one day and down to 102.3° F. the following day. The animal died. From the clinical symptoms noted, the immediate cause of death was pneumonia. The pathological findings of this case will be discussed later. In this case the pulse would vary according to the temperature. The animal would take food only at intervals of days. One case of which we spoke before, that had the purulent dermatitis, showed permanent derangement of equilibrium before pneumonia prostrated him. We are unable to determine whether the cause lies in the central nervous system or in the semicircular canals. The animal, while up, would walk or sidle to the left, with the legs bracing the body as on a sawhorse. He would prop himself against the walls and rotate to the right. The head was drawn to the left. After he went into prostration, the head was still drawn to the left. The animal also had incontinence of urine. Autopsy showed occlusion of the semicircular canals; the fluid was solid or gelatinous. This condition of the canals seems to be responsible for the loss of equilibrium.

Treatment

The treatment of the disease has been purely symptomatic; phytolacca and sommos, for quieting; luminol and paralga, as sedatives; calcidin; triple arsenates with nuclein; fraisses hypodermatically; ice-packs on the head in extreme cases; fibrogen hypodermatically, to check diarrhea and hemorrhages; bismuth and salol and weak solutions of silver nitrate, as a stomach lavage or in tablet form. We think that a vaccine or serum can be made, both as a preventive and a cure, as soon as enough research work has been done.

Owing to the fact that mercurochrome has come to universal use in the medical profession, especially in the treatment of infants, we considered it in the treatment of this disease. We know that mercurochrome is very beneficial in reducing temperatures and bringing on an early crisis, which is most gratifying in results. With this knowledge at hand, we attempted it in the treatment of this malady with very satisfactory results. We use this drug, ¼ to ½ of 1 per cent solution intravenously, in dosages of from 3 to 10 cc, according to the size and weight of the subject being treated. On dogs from 40 to 60 pounds, 10 cc were given. In this treatment, the jugular vein was utilized, it being the largest vein and the safest. Ordinary aseptic precautions were used. We were exceedingly careful not to get the medicine outside of the vein, as a sterile abscess would result. Difficulty was experienced in using the smaller veins, as the mercurochrome would contract the walls of the veins when injected and the veins would not take it up rapidly. Results were gratifying, as in all cases the temperatures were lowered in from 12 to 24 hours. One to three doses were generally given, three to five days apart. In one case, it required only one dose of 6 cc to allay all fits. No other treatment except several doses of somnos, to allay convulsions, and metagen compound were used. We have used this treatment on twenty-one cases and are very well pleased with results. It is worth your trial. We always recommend the simultaneous distemper prophylaxis to avoid distemper complications.

Diet

In the milder form, regular table diet is advisable, such as meat, bread and vegetables mixed together. In severe cases, we give predigested beef, raw eggs, milk, broth and, sometimes, liquid peptonoids; also barley meal in boiled water.

Pathology

Twenty-two autopsies were made of this disease.

External examination: Fourteen of the animals showed great emaciation; the rest were in fair condition. In all of the cases, the eyes were sunken, the facial muscles atrophied, and the conjunctiva catarrhal and hyperemic. The buccal membranes in all cases were bluish in appearance, the buccal mucosa of nine showed lacerations; the tongues in those cases also were lacerated.

Internal examination: All of the subjects showed normal positions of the organs except in two cases. These two showed a disarrangement of the intestines, the spleen being farther posterior than normal. This condition can probably be explained by powerful contractions of the intestines or by the dog throwing himself about while in a fit. The abdominal peritoneum in all of the cases except eight was shiny and transparent. Eight showed subperitoneal hemorrhages and hyperemia. These eight also showed about 40 cc of sanguineous yellow fluid in the abdominal cavity.

Kidneys: In all of the cases, the kidneys were normal in size. The capsules were shiny and transparent. They peeled easily, showing an acute inflammation. In chronic inflammation, the capsules would be adherent, due to an increase in interstitial connective tissue. On section, all of the kidneys bulged. The cortex was darker than normal. Lines of darker and lighter areas were noticed in the cortex. This condition indicated hyperemia of the vessels in the interstitial connective tissue and degeneration of the epithelium of the kidney tubules. The periphery of the medulla was pale. The calyx and hilus contained shiny viscid material that was catarrhal in nature. In three cases, blood was found in the hilus. On pressing the kidney tissue between the finger and thumb, the tissue was soft, indicating an acute process.

Adrenals: In all cases but three, the adrenals were normal. These three were slightly enlarged, bulged on section and were edematous.

Intestines: In all cases, the rectum and colon contained only slight amounts of fecal material, this being shiny and fetid in nature, often streaked with blood. The mucous membranes in all cases were catarrhal and showed hemorrhages and hyperemia. In most cases, the cecum was impacted with a semi-solid mass of fecal matter. The mucosa in all cases was catarrhal. The contents of the ileum were semi-solid and, for the most part, yellow due to eggs being used as food material. The vessels of the mesentery were injected. In four cases, the mucosa was mucopurulent. Hemorrhages of extravasation type were seen in the mucosa. The eighteen other cases showed a catarrhal condition with petechiae and ecchymoses. The contents of the jejunum in all cases were more fluid in form and mixed with mucus. The mucosae in all cases were dull, due to a catarrhal condition. Four of these showed a mucopurulent exudate with

ulcers. In these four cases hookworms were present. The contents of the duodenum were shiny in consistency. In eighteen cases a catarrhal condition was noted, with hemorrhages in the mucosa. Four cases showed a mucopurulent condition. Peyer's patches were elevated, showing an inflammatory process. The mesenteric lymph-glands were enlarged and edematous in all cases.

Liver: In all cases, the liver was enlarged, darker than normal, for the most part. One liver was so large that it ruptured in three places. On section, blood oozed from the liver in all cases. The liver tissue was very friable; the bile-ducts contained bile and a catarrhal condition was noted. The gall-bladder was always distended with dark-colored bile. In one case the bile was thick in consistency.

Pancreas: Normal in all cases.

Spleen: The spleen in all cases was enlarged to a greater or lesser extent. The spleen was soft and, in five cases, friable. The splenic capsules peeled easily. On section, the trabeculae were visible and the pulp was mushy in consistency. In five cases hemorrhages were noted beneath the splenic capsule.

Pleural cavity: The pleura was shiny in all cases except five. These five cases showed subpleural hemorrhages and hyperemia. These cases also showed a sanguineous, yellow exudate in the pleural cavity.

Lungs: Emphysema was seen in nine cases. The lungs in all cases were hyperemic and, in five cases, showed hemorrhages. The bronchi and trachea showed a catarrhal exudate which, in some cases, involved the peribronchial lung tissue.

Heart and pericardial sac: The pericardial sac contained a slight increase in pericardial fluid. The coronary vessels of the heart were slightly injected. The epicardium in all cases appeared normal; myocardium, normal; endocardium, normal.

Brain and spinal cord: The meninges in all cases were thickened and the vessels hyperemic. Submeningeal hemorrhages were seen in every case. Perivascular hemorrhages were seen in every case. Perivascular hemorrhages were seen around the arteries on the surface of the brain. On section, petechiae and ecchymoses were seen throughout the nervous tissue. The lateral ventricles in all cases were distended with fluid of a sanguineous character and there was fluid beneath the meninges.

Blood: No parasites were present in the blood; neither were any bacteria encountered. In those cases where intestinal

parasites were present, there was an increase in eosinophiles. Normally, eosinophiles range from 1 to 6½ per cent. Our results showed from 3 to 5 per cent in those cases free from parasites and in those cases with parasites, from 7 to 11 per cent. The hemoglobin content showed from 40 to 60 per cent, whereas the normal is about 90 per cent. The erythrocytes were lower than normal in all cases. In some of our more severe cases, the erythrocyte count was as low as 2,500,000. Our leucocyte count was a great deal higher than normal. In one particular case, the count was made just before the animal had a fit. The leucocyte count was 15,540. After the fit, another count was made and found to be 16,302. In one case, the leucocyte count was 23,000. Three checks were run on each count to insure accuracy. In getting the differential count, it was noticed that the polymorphs and lymphocytes showed the greatest increase, the average being 73 per cent. Lymphocytes, 20 per cent; large mononuclears, 6 per cent; eosinophiles, ½ of 1 per cent, except in these cases where the intestinal parasites were noted. The decrease in red cells brought into the picture of the blood, macrocytes, megalocytes and poikilocytes. Nucleated red cells were noted in one dog, with normoblasts, microblasts and megaloblasts.

Bacteriological Investigations

In three dogs we found the same organism, a short bacillus, in the spinal fluid, spleen and liver. On artificial media, it grows luxuriantly. It produces acid and gas on dextrose, lactose and saccharose. It is Gram-negative. On inoculation, the organism threw guinea pigs into convulsions in four hours and killed them in fourteen hours. The injections were made into the peritoneal cavity, 1 cc being injected. On autopsy, the sanguineous, yellow fluid in the abdominal cavities of the pigs contained the organism injected and it was re-injected into dogs. One dog was gven 1 cc intraperitoneally; the other dog subcutaneously. These dogs showed nervousness and fits as early as fifteen hours after injection. The dog injected in the peritoneal cavity died in three days, in convulsions; the dog injected subcutaneously showed a slight nervousness, but recovered. Autopsy of the first dog injected showed typical symptoms with marked hemorrhagic gastro-enterities and peritonitis. Some of the spinal fluid from one of the dogs was passed through a Berkefeld filter and injected into pigs with negative results. In all cases where

organisms were found, they were recovered from the experiment animals on death.

Natural Infection

In order to find more out about the disease, we ran an experiment on three puppies in the hospital to see if the disease is contagious. The pups were all fed the same food material. One of the pups was allowed to go where he pleased in the hospital and to associate with all frights disease cases. This pup (1) was the largest and thriftiest of the three. Pup 2 was allowed to associate both with its mother and pup 1, but was not allowed to associate with any of the patients. Pup 3 was fed artificially and used as a control. Pup 1 showed no signs of the disease for about twelve days. At that time, be became languid and refused food. Three days later, he began to have fits, first light, then increasing in severity, as the disease progressed. The animal died in a few days. Pups 2 and 3 were under our observation for 36 days and during that time showed no signs of the disease. On autopsy of pup 1, the lesions were diagnostic. The blood before death was also diagnostic. This experiment is in no way conclusive and we do not want to convey that idea. It is merely to show you what we have done and the results obtained.

Additional Pathology

On eleven autopsies, the same lesions were noted in all cases except four. In two of these cases there was a purulent dermatitis with ulcerative keratitis. A staphylococcus was the causative factor in the dermatitis. Staphylomas were seen on both eyes in both dogs. In one case, atrophy of the optic nerves was noted as spoken of before. The one outstanding case was a male setter. The organs were normal in position. Large gangrenous infarcts were seen in the kidneys. The urocyst showed a catarrhal inflammation. The intestines did not contain any worms, and gangrenous areas were seen throughout. The liver showed gangrenous areas also. The spleen was swollen, darker than normal and was very soft. The lungs were gangrenous throughout and were almost completely solidified. There was also about 15 cc of foul-smelling exudate in both the pleural cavity and the abdominal cavity.

Two cases that had jaundice showed the following lesions: All organs and musculature were as yellow as gold. Other lesions were not pronounced as in the uncomplicated cases, except for the intestinal tract. Urine examination, by the Hay's

method, showed the presence of bile. Urine examination was made in thirty-four cases and albumen was present in sufficient quantity to be noted easily in twenty-one. In the other cases, the reaction was doubtful. Sugar was noted in nineteen cases. The specific grivity varied so much that we could not get anything of any value from it. Microscopical examination of the urine showed all kinds of bacteria; also epithelial cells and pus cells. Blood examinations were carried on but nothing new was noted.

Etiology

Some people have attributed the cause of this disease to diet. From 183 cases on our records of recent months, we have collected the following data on diet: Some 138 have been fed on table scraps, 21 on cornbread and meat, the rest on dog food. From variance in diet and under varying conditions of health and breeds, we would like to know how diet could cause the disease. If the disease is caused by food poisons, would all foods produce the same kind of toxemia?

From our work, we can not see how worms cause the disease for, in 22 autopsies, only four showed the presence of worms. Fecal examinations of all the cases showed that only a very few showed the presence of worms. In most cases, the owners had wormed and re-wormed the dogs, thinking that worms were the cause of the disease. If worms cause it, or throw off toxins which would cause it, why did they not produce it fifty years ago? Have they changed morphologically or physiologically since then?

Some have claimed it to be constitutional, but our observations on all breeds show that none is immune. It would seem that the ones most affected are the ones that run out of doors. Pet dogs continually kept in the house have the disease, but our figures do not show the proportion to be as great as compared with those that run out of doors.

In conclusion, our idea is that it is an infectious and perhaps a contagious disease, from some organism or the toxin that it produces, which affects the central nervous system and causes disturbances in the other functional organs.

Discussion

Dr. J. W. Scheibler: In what little experience I have had with this disease, I have treated some dogs for internal parasites, as Dr. Wright has said, and I have changed the diet. I have seen good results follow. After taking the dogs off starchy foods, such as cake, candy and potatoes, they have shown marked recoveries in about two or three weeks. Then, of course, I

have had other dogs that would go from bad to worse. These dogs I have given quinin, nux vomica and Fowler's solution, and sedatives, such as three grains of chloralhydrate, every two to three hours.

DR. F. P. CAUGHMAN: Is there any difference between the fits we see accompanying distemper and the fits that you have described in these young dogs?

DR. WRIGHT: After these dogs get down in a semi-comatose state, the fits resemble distemper fits quite a bit. We have had dogs in the hospital that had recovered from distemper and they came down with running fits in from one to three years after they had distemper. That is the reason we believe that it is not distemper.

MEMBER: Is fright disease just as prevalent in dogs that have not had canine distemper as in those that have had it?

DR. WRIGHT: Puppies and old dogs apparently in good shape, in good flesh and with no symptoms whatever, would come down all at once. The first symptom they would show lots of times would be to start barking and running, as if a yellow-jacket or something was stinging them. Generally the woman gets hysterical and yells that the dog is going mad, but it is so prevalent now that people know they are not mad and do not shoot them. Some people say they will not bite, but they will. I caught one the other day and it bit me. They will bite when they are having a fit.

DR. A. J. STEINER: I possibly got the same impression in differentiating between these fits. If everything Dr. Wright describes is fits, I do not know why we should call them fright disease. We have an awful lot of fits here and probably lots of you have had the condition where the dog will start out and run and howl, but I do not consider that fright disease. Lots of times they want you to go out and watch a dog to see if you can not determine the cause. In some of those cases I can not help but believe that it is hookworms. I have given them hookworm treatment and they have not had any more fits. I would not say hookworms cause it, however.

MEMBER: I would like to know if everything we call fits is fright disease.

DR. WRIGHT: We call them fits, as a common term. They run themselves until they become exhausted and fall. I think that is brought on by the congested condition of the brain. Loud talking will do it. Puppies while eating will rattle their pans. Unnecessary racket will bring it on; shake a newspaper and that will bring it on. You can call it a fit or call it excitability, but when they run until they exhaust themselves and then get down and kick and slobber and do everything else, that is what I call a common fit, which we think is caused by this organism.

DR. O. M. NORTON: If I understood you correctly, you said you had an ulceration of the cornea.

DR. WRIGHT: In a few cases.

DR. NORTON: I think you have two or three diseases confused. You have your complications, including conditions of the eye, and the disturbance of your blood circulation, but it seems to me, where you have ulceration of the eye, which we have in the South a great deal, that is a disease in itself and has no connection with running fits. I have seen dogs in the South with so-called running fits, for twelve years or longer. Of course, in recent years, it looks as if it has become more general and passed to other localities. The first cases I saw, about twelve or fifteen years ago, were in town and were fed entirely on cornbread. Dogs eat cornbread only down there. At that time I changed the diet on these dogs and stopped that. It is a peculiar thing. I believe we have the disease—running fits— but I believe this, that lots of so-called cases of running fits are complications of distemper and also complications of worms. Anything that irritates the intestinal tract brings on running fits.

A few years ago, we had a meeting of the Tri-State Association, at Memphis. Dr. Flynn, I think, was down there and he seemed to think that it was due to the feeding. I did not see it at that time, and I can not see it at the present time. We get dogs that are fed everything, some correctly and some just a ration of cornbread. It does not seem to make much difference what the feed is. The treatment that we have been using, and with success, is bromid of valerian. The first three days we give a dose three or four times a day, sometimes five times a day, for a bad case. Then we shut down to about three times a day for three days and then two times a day for three more days and

then run off to once a day. If a dog has a history of worms, we give him worm treatment before we start.

DR. WRIGHT: We make an examination of the feces in all our cases and find that not over four per cent of our cases of running fits have any intestinal parasites at all.

DR. NORTON: We thought hookworms had a good deal to do with it at the start, but we have found out that this is not always the case. If we did not get results from hookworm treatment first, we followed with the tapeworm treatment, and then possibly with the other one. In lots of these dogs, we failed the first time with the hookworm treatment but got results afterwards with the other.

DR. WRIGHT: On autopsy, as I stated in the paper, only a very few of these dogs show any intestinal parasites whatever, hookworms, tapeworms, or any kind.

DR. FLYNN: Dr. Norton quoted me as saying that all fits were due to dietary disturbance. I do not hold that view.

DR. NORTON: I meant to say that you said at one time that you thought that the feed was the primary cause; that certain dog foods caused fits in dogs and that your idea was that it was due to an unbalanced ration or feeding improperly.

DR. FLYNN: I want to make this statement so as to get it clear in the minds of everyone here and that is that I do not believe that all fits are caused from dietary disturbance. Fits may result from distemper or may be caused by intestinal parasites, or heat, or teething, or a combination of these causes. I make the statement this way—that true fright disease, as I understand it, is a layman's term for a certain running fit. The first I heard of the condition was down in the southern states and I contend that the condition that they refer to as fright disease is not a fatal disease and it is due to the diet and can be produced by improper diet, but I would not want the profession to get the idea that I thought that all forms of hysteria are fright disease or are caused by improper diet.

DR. WRIGHT: Doctor, let me ask you a question. You came by Atlanta, several years ago, and at that time you thought it was caused by dog biscuit. You said you got a sack of dog bread down below Kansas City somewhere and produced the disease with dog biscuits. We have thousands of dogs in the South that never saw a biscuit. We have records of around two or three hundred cases that have been fed all kinds of diet from the richest down. Now if diet causes that disease, how is it that we can take the fluid out of a dog and inject it in a guinea pig and throw the pig into typical convulsions? And then we take the abdominal fluid out of that guinea pig, put it into a dog and produce a typical barking fit in fifteen hours and the dog dies in four days. The one injected simultaneously did not throw the running or barking fit, but was very nervous.

DR. E. C. PORTER: I would like to ask what percentage of your dogs were fed an entirely meat diet?

DR. WRIGHT: I think twelve out of 250.

DR. G. W. LITTLE: We have a different condition in the East. I think these boys from the East will agree with me that most cases of so-called fright disease are simply complications of a worm infestation, of the tapeworm, hookworm, whipworm, often combined in the same individual, or the hookworm alone or the whipworm alone. Now then, I will cite one case where the fright condition was caused by whipworms alone. A dog would start running and run for quite a distance, falling into a convulsion. Fecal examination revealed the presence of the whipworms without any of the other parasites. We removed the cecum and the dog increased in weight and general condition but still he had, more or less frequently, these attacks of running fits and we were still able to find the whipworm eggs in the feces, which proved beyond the shadow of a doubt that the whipworms were living in the main bowel, as well as the cecum. Hall said that in a certain small percentage of cases the whipworms did live in the main bowel as well as the cecum. Now then, in the East, as far as my practice is concerned, we have never seen a case of this condition that was not complicated by a worm infestation and I feel that Dr. Wright's material and postmortem here is a revelation to most

of us in the East and really substantiates the fact that we have a disease which is apart and separate from those caused by internal parasites or distemper.

DR. WRIGHT: We all know that we have worm fits; we had them before we had so-called fright disease. A famous bird-dog man told me about operations that you and another veterinarian in New York were doing in these whipworm cases. Well, Dr. Rife and I went back and we looked for whipworms in all of our autopsies. We recorded every parasite of any description that we found. We found only about four per cent in numerous autopsies, I think 112, in two or three years. We have a dog in the hospital now that apparently, from no cause whatever, developed a fit and went as blind as a bat and is still blind and has as normal an eye as you ever looked into. You would not know he was blind except when he puts his nose on the ground and feels along. We not only have one but several.

DR. H. W. BROWN: To substantiate what Dr. Flynn says, I happened to be associated with him at the time of the experiment with the biscuit, on running fits. We had a letter from a man in Oklahoma who had twenty-three beagle-hounds and out of the twenty-three, seventeen of them were having these running fits and we sent for some of his diet. He sent us the dog biscuit and we fed it to two dogs and in five days produced the fits, a type I call running fits, with no complications whatever. The dog would bark and yell and howl and in a few minutes was over it and perfectly normal again. We gave him a good cathartic and changed the diet to raw meat and buttermilk and the dog was apparently normal within a week. We let him go for about two weeks and put him back on the dog biscuit and within five days we reproduced the fits again. That same biscuit was shipped to Indiana, to the Indiana Veterinary College. We fed it to two dogs there and produced the fits in two dogs that had never been fed that particular biscuit. We had that biscuit analyzed and could find absolutely nothing to cause the trouble.

Then I find that in different localities in practice, in Kansas City, and last year in Portland, Oregon, running fits are different, but in Indiana, so far as my experience has been, I have had no fatalities and I have made it a special point to find out in the locality in which I am located now, in northern Indiana, that practically every dog that comes to my hospital with running fits, with one exception, has been fed on sweet milk. I do not think that sweet milk causes the fits at all, but I do think that diet and teething disorders have more to do with it than other things, because those dogs have been given a cathartic, not necessarily wormed in every case, but some of them were wormed and some not. I have had them recover by changing the diet to raw meat with buttermilk. I have had any number of them recover without any treatment whatever.

I have a dog in my hospital now that belongs to me, an Irish setter, that had distemper when three months old. He had one fit a month ever since, until one month ago. His is now seventeen months old. He had a typical running fit, with no complications whatever, a short time ago, and I wormed him. I got a few tapeworms. Following that I had so much trouble with his whining at night that one night I thought I would get a little rest and I gave him three grains of morphin, put him to sleep and the dog has never had a fit since and has never shown any nervousness since. I do not think that is a specific. I do not think anything is a specific, because in any cases that I have had, in my experience I mean, I have found a number of causes combined: teething, worms, dietetic disorders, and all those things. I have never been able to reproduce the disease by putting dogs in contact.

DR. C. C. RIFE: In making our bacteriological examinations, we kill the dog in the agonal stage, before the organisms from the intestinal tract invade the other organs of the body. In four or five cases we have taken dogs, put them to sleep and made a spinal puncture, in the region of the neck. We have also punctured the cerebrum and have drawn off the fluid that way. As far as worms are concerned, we record whether any worms are found in all dogs that come to the hospital with fits. We check up on them and find that few of them have worms.

DR. HAIGLER: I would like to ask Dr. Wright if he finds any appreciable percentage of these cases developing distemper in one to two to three weeks after fright disease.

DR. WRIGHT: If they are real young dogs we have ten per cent that break down with distemper inside of three weeks. If they are old dogs, they do not have it.

DR. HAIGLER: What is the mortality?

DR. WRIGHT: We cure 75 per cent of them. We often have a dog that does not have but one fit.

DR. HAIGLER: I believe Dr. Lacroix has said that he believes that practically all cases of so-called fright disease will develop into distemper in from one to four weeks. I think I have seen his statement and I just wondered if there is any difference in the locality or if you have had the same experience.

DR. WRIGHT: I explained that a while ago. To remove the benefit of a doubt that it was distemper causing it, we have lots of cases where they have had distemper one to three years before and then came down with these typical fits.

DR. C. R. WALTER: When a dog runs for half an hour or so, or four hours, and you autopsy this dog, do you not find hyperemia of the brain caused from nothing else than the temperature, like a case of overheating in a horse? We at Tulsa have so many running fits that we almost have them ourselves sometimes and we attribute the cause to improper diet, parasites and distemper.

DR. WRIGHT: Doctor, you get a hyperemia on any animal, or yourself, if you have your skull cap lifted off, but it does not seem that you would have a hemorrhage there, for if you did every race horse, dog or anything that runs would have. I think that what brings on this fit is a hemorrhage at the start. Now, how in the world, will anybody tell me, can you produce the typical symptoms with a hypodermic needle, if it is caused from dietetic conditions?

DR. BROWN: Why is it then that we have so many cases, when clients come to your hospital, the minute you set a pan of food in front of these dogs, they will throw one of those fits? There is no excitability at the time. If they are overfed that should not excite them.

DR. WRIGHT: Excitability. That shows that there must be a toxin in that dog's system or he could not bring it on him that way.

DR. MILKS: I do not know anything about this. We have seen what we call running disease in the North, but I have come to the conclusion that when we speak of running disease or a convulsion up there, we are treating a symptom instead of a real thing. Now, if you stop to think, I would like to know how many of you can tell us what a convulsion is. Why is one? You will find by looking it up that nobody knows the why of a convulsion. The nearest we can get to it is a spreading of the reflexes, so that our nerve impulses spread through channels not ordinarily involved. We have seen these cases and when I say we incriminate a certain thing, and the disease clears up, we think that is the cause of it. We have seen this commonly in all kinds of parasitic infestations of the intestinal tract. We have seen it in distemper and we have seen it in rickets. Rickets is quite a common cause of convulsions. We have seen it in indigestion and I think certain foods do it sometimes. A certain factor is working against it. When one overcomes the other, we get the response or the bursting of the nervous energy. There is another disease that has not been mentioned, that we see quite commonly as the cause of running fits. It is nothing new to us. The hookworm we find is a frequent cause. Then we find, on top of that, some dogs get bewildered, excited. I remember a neighbor of mine had one of those dogs have a fit in a department store. I did not see it, but another veterinarian gave the dog a dose of morphin and that was the end of it.

Then I wonder sometimes, if we are not dealing with true epilepsy. Again I think like all of you, we have those cases we can not attribute to anything. I think I mentioned distemper. We find it very common in the beginning of distemper in young dogs. I know I have not enumerated or covered the ground so as to clear the thing up; I am simply citing our experiences with it. I am not familiar with the disease in any other place except around Ithaca and I do know we have been able to clear it up by treating for parasites, so that when we get one of those dogs, the first thing we do we make a fecal examination and clean it up and that is usually the end of it. Sometimes distemper comes on. We have had numerous cases where it was caused by parasites. I remember distinctly, a pack of ten pointers having these spells

and every last one of them had a bad infestation of ear parasites. When we cleaned them up, that was the end of it. So I think we have a symptom instead of a disease and when we get the case it is necessary to hunt around a little to see what we can get.

DR. HAMLET MOORE: My experience with running fits dates back quite a little time. I first saw it in pets that have been harbored in hotels or apartment houses and babied along, then taken out for a ride to the park or square and turned loose in the heat of the day. I thought that was simply all there was to it and all of those dogs I treated in the same manner. The minute a dog would come to me, no matter what they had done to him, I would give him a grain of morhpin and forget about him. After the dog had worn off the morphin, I always gave the dog, no matter that kind he was, what I term a digestive, consisting of equal parts of heavy oxid of magnesia and bicarbonate of soda. That, given in a little sweetened water, making it palatable for the dog, is preferable. In 85 per cent of the cases they will evacuate the contents of the stomach in five to twelve minutes. Until time grew on—and I believe that has been everybody's experience—I believe that running fits seemed to arise from no apparent cause and, if there is anybody who knows anything about it, he knows a certain adjective more than I do.

MORE COUNTIES FREED OF BOVINE TUBERCULOSIS

An official order of the Bureau of Animal Industry, U. S. Department of Agriculture, recently added four counties and several parts of counties to the extensive area already freed from bovine tuberculosis. The new counties are Knox County, Indiana; Hartnett County, North Carolina; Lawrence County, Pennsylvania; and Shelby County, Tennessee. Besides these new areas the government recognizes also, as modified accredited areas, parts of three counties in the state of Vermont. The areas which have earned this recognition are that part of Washington County included in the town of Berlin, the part of Lamoille County included in the town of Johnson, and the part of Caledonia County included in the town of Peacham.

The bureau has also reaccredited other areas for an additional period of three years, following completion of necessary tests. The reaccredited areas are Starly and Stokes counties in North Carolina, and Ohio County in Indiana.

To obtain the recognition mentioned, the cattle of an area must be tested for tuberculosis by a State or Federal veterinarian and the result of the test must show not more than one-half of one per cent reactors, such animals, if any, being promptly disposed of by slaughter. The total number of counties in the United States on the modified accredited list is now 306.

If you can find a single housefly in Alaska, you'll do better—or worse—than National Museum investigators who have been up there trying to.—*Farm and Fireside.*

BLACK TONGUE*

By C. C. Rife, *Atlanta, Ga.*

Black tongue or dog typhus, as defined by different authorities, is an acute infectious disease, characterized by a severe gastro-enteritis and stomatitis, and occurring in epizoötic form.

Etiology

The etiology of this disease has been in question for years and different men differ as to what the causative agent might be. Some claim the disease to be due to a specific organism. Some few believe it to be due to a constitutional derangement. In passing, I will state what several men did on this disease, in order to refresh your minds. Horning claims the disease is due to a toxemia, caused by toxins from the colon bacillus. He also conducted experiments in which intravenous injections of the colon bacillus produced a severe gastro-enteritis. We did the same thing on one dog and got a severe gastro-enteritis, with vomiting, purging, passing of blood, etc., but with no typical symptoms of black tongue. Others have found organisms in the blood, belonging to the Pasteurella group, but none have produced the disease in the typical form.

Symptoms

The disease that we describe as black tongue has symptoms that differ considerably in some respects from those recorded by authors of text-books and articles. It is very seldom that we see dogs with this disease in the initial stages, which probably alters the picture somewhat. We have, in the past year, had the opportunity of handling five cases from the onset, with the symptoms of inappetence, but with little or no vomiting. On asking our clients for the history, they often said that the dog had refused to eat and some claimed the animals had vomited, while others said they had not. It is seldom that we get a case where the animal is suffering from constipation.

When we get a case of black tongue, the patient is almost always in the second stage of the disease and has the following symptoms: He is generally in good or fair condition of flesh; hair-coat is generally rough; skin, dry; ropy, slimy, foul-smelling, tenacious saliva drools from the mouth and coats the hair on the

*Presented at the sixty-third annual meeting of the American Veterinary Medical Association, Lexington, Kentucky, August 17-20, 1926.

fore legs and around the mouth. This saliva is generally gray in color and, if the animal has taken any food previous to our seeing him, is frequently mixed with food particles. The nose is generally dry and parched. The temperature in most cases is normal or subnormal. Occasionally, the temperature will be up a degree or so. Respirations are generally normal at this stage of the disease. Pulse is a little fast and weak. As the disease progresses, the animal shows complete anorexia. On the other hand, he will drink water, of which we give only small quantities, as it produces vomiting. The bloody diarrhea starts in this stage and is generally uncontrollable.

It is in the second stage that we see the greatest change in the mouth. In those initial stages that we have seen, the gums, buccal mucosa and tongue are generally congested. In the second stage, areas of irregular shape and size begin to turn gray on the mucous membrane around the margins of the teeth, along the borders of the tongue, and on the buccal mucosa. The tongue at this stage becomes dark red to blue and the mucosa a deeper red where the necrotic patches have not made their appearance. Often the dorsum of the tongue becomes coated and shiny. Then the exudate from the mouth may be stained with blood. The necrotic patches increase and often become confluent. As the necrotic membranes are washed off in treating, angry, deep-seated, red ulcers remain. This is about the third to the fifth day of the disease and, from then on, the animal becomes emaciated and weak, walking with a staggering gait. At this stage, the pupils of the eyes may be dilated slightly and the conjunctivae hyperemic. Often a little pus may be seen in the eyes in the morning. The eyes are generally sunken.

Respirations are generally accelerated; temperature, 97 to 99° F.; pulse, fast and weak. The skin becomes doughy, indicating a weak heart and bad circulation. Extremities become cold. The animal voids only a little urine, this generally being highly colored and strong. The animal does not seem to be in any pain. On handling, some may show pain, due to pressure on the abdomen, etc. About the sixth to the eighth day, if the animal does not show improvement, he grows weaker rapidly, with bloody fecal matter passing from the bowels involuntarily.

Pathology

All dogs that died of black tongue were autopsied. The pathology of these cases varies a great deal, due to the course

of the disease. The skin and hair-coat are rough and dry; the skin is loose and in folds, due to great emaciation. The longer the disease lasts, the poorer the condition of the animal's body. The eyes are sunken and the mucous membranes pale, due to loss of blood through hemorrhages of the bowels. In those cases where hemorrhages from the bowels have not been profuse, the membranes are generally hyperemic. Occasionally, an exudate will be found in the eyes. The oral cavity shows extensive areas of necrosis and ulcerations. The tongue will be cyanotic and contracted, and the borders will often contain ulcers. The abdominal peritoneum is generally shiny. Visceral peritoneum almost always shows some inflammation, the mesenteric vessels being injected and the lymph-glands swollen. The kidneys are generally hyperemic and occasionally hemorrhages will be found. The adrenals are generally swollen and edematous. In three autopsies, I found hemorrhages in the pancreas. All livers I have seen were swollen, hyperemic, and some show hemorrhages. Gall-bladders are generally distended with bile.

In making eighteen autopsies consecutively, only three cases were found in which they coincided with what is written in text-books about lesions in the intestinal tracts. In the other fifteen cases, the small intestines and stomach showed acute inflammation but no hemorrhages, except for a few petechiae. The mucous membranes are swollen. I also noted necrotic nodules throughout the intestines in all cases, being more numerous in the large colon. The esophagus was hyperemic and catarrhal, as was the stomach. The pharynx showed large necrotic areas and hyperemia, where the animals had not bled so profusely from the bowels.

Two cases showed pneumonia. Other cases showed no lesions except a little edema. The heart was nearly always pale and streaked. The nervous system showed inflammation in some cases but, in most cases, no change could be noted except for hyperemia. Examinations of the urocyst showed the organ contracted in nearly all cases. The mucosa showed petechiae in some instances. Examination of the urine showed a high specific gravity (4 plus), indicanuria and albuminuria; acid, high.

Diagnosis

In most cases, diagnosis is easily made from the lesions of the mouth and from the temperature. The odor is also typical, together with the ropy saliva. The dogs are nearly always

quiet and lethargic. Occasionally, some difficulty is experienced in making a diagnosis between black tongue and poisoning from the government rat poison. Often the lesions in the mouth are practically the same. The high temperature (104° to 106° F.) in poisoning will aid in making the diagnosis. Also, the pupils of the eyes are dilated more in poisoning than in black tongue.

In both conditions the animal salivates profusely; the mucous membranes of the eyes are injected in both. In poisoning, the animal is in great pain and it is evidenced by continuous howling. If the diagnosis is not made correctly and the right treatment used, the animals will soon die. The short duration of the disease will also aid in diagnosing. One may get confused with ulcerative stomatitis, but remember that the temperature is generally up in this disease and the condition comes on slowly. The lymph-glands in the region of the throat and neck also become enlarged in ulcerative stomatitis, but you never see this condition in black tongue. You never see vomiting and gastro-enteritis in ulcerative stomatitis.

Course

The course of the disease is generally from five to ten days. Usually, the course is drawn out over a week and seldom over fourteen days. We have had cases to die within three days, but when we received them, their condition was poor and the disease was in its severest form. Prognosis is always grave and you can expect to lose from 60 to 75 per cent.

Clinical Observations

During 1925, we treated fifty-six cases of black tongue in our hospital, on which we have records. Of these fifty-six cases, we cured seventeen; thirty-nine died, giving a mortality rate of 69.5 per cent. So far in 1926, we have treated twenty-six cases, nine of which recovered, the other seventeen died. This gives us a mortality rate of 65.3 per cent. You can see from these figures that the prognosis is grave and if we save two dogs in succession, we feel highly elated. We also compiled some figures on the frequency of the disease in the different breeds of dogs, and found that pointers and setters headed the list and hounds were second, collies came third and bulldogs next. It also might be interesting to know that the heaviest mortality comes in the hunting breeds. It seems that house pets are the least affected by this disease and more readily cured. It is the high percentage of recoveries in these breeds that brings up the total percentage

of those cured. I also noted what months of the year in which we have the most cases of black tongue. March, April, May, September and October stand out with the most cases.

Some claim the disease to be due to a specific organism, but we have conducted experiments and have been unable to transmit the disease. We have tried feeding experiments, but in no way could we infect any animals. Bacteriological examinations of ulcers, upon culture in nearly all cases show a staphylococcus infection. This organism is probably a secondary invader. Fecal examinations showed hookworm eggs in a large number of cases. Bacteriological examinations of the feces shows the presence of the *Bacillus coli communis* and *Bacillus coli communior*. We also made blood examinations and found a mild leucocytosis. The blood was cultured, but always found sterile.

Treatment

Our first thing in treatment is to take all the water away from the animals, except for a little crushed ice, two or three times a day. If water is given, the animals are liable to vomit, and this we try to prevent. Then we withhold all food for two or three days, as the stomach is in such a condition that food can not be assimilated. We found this out through autopsies. When we start feeding, we give raw eggs with predigested beef bouillon. Often, after fasting two or three days, the dog can not assimilate this and starts vomiting, thereby becoming very much weaker by the act of vomiting.

We also treat the local symptoms of the mouth. We have used different drugs in the treatment of the mouth. Acriflavine and proflavine (1 to 1000), as a mouth-wash or swab, every three hours, has been found beneficial. Mercurochrome in the same strength has also worked well in some cases. Blue stone and vinegar are good. All of these drugs are good for mouth washes and aid the healing of necrotic patches. For the intestinal symptoms, we have tried trypacrin-B (Jen-Sal) for intraperitoneal injection. On several cases we had good results, but our luck did not last long. On autopsies, after the use of this drug, we found a peritonitis which was not due to any organism, as the exudate was cultured and found to be sterile. Our conclusions were that this drug was not taken up as it should have been, so we discontinued its use.

We also used a 1-to-1000 solution of neutral acriflavine and proflavine intravenously. We saved several dogs by this treatment, but also lost some. With these injections we also gave

bismuth and salol, every two hours, to check diarrhea. Fibrosol was tried, to check diarrhea, but with little or no effect. Adrenalin chlorid was also tried, with slightly better results, in checking hemorrhage. Adrenalin in such cases is advantageous, because it fortifies the heart, which is weak at this stage of the disease. Camphosol (1 to 3 cc) is used to fortify the heart in bad cases. In those cases where pneumonia symptoms occur, we treat them by the use of camphorated jackets. Golden seal and bismuth subnitrate, gr. v, t. i. d., have been used with varying results.

With any of the above drugs you may get a treatment that will cure some, while others receiving the same treatment will die. We feel that good, common-sense treatment, plenty of nursing, together with nourishment when the dog can take it, will save all that can be saved until the cause of the disease is found.

Conclusions

If the disease is constitutional, as some believe it to be, the medical treatment of symptoms will not do a great deal toward saving dogs. Green, of Minnesota, claims the disease to be constitutional and claims it can be treated successfully by feeding carrots and vegetables. We had one dog in our care that developed the disease four times in one year. If the disease is bacterial in nature, it would seem that there would be an immunity established, which at least would be temporary. Again, the hunting dogs are the ones most generally affected and their diet is generally meat and corn bread, whereas the house dog gets a more varied diet. From this fact it would be possible that the disease is caused by some deficiency in diet, probably some of the amino-acids being lacking. We hope this point will be worked out later. I feel confident that it will be a deficiency of some kind, because all the facts point that way and not toward an infection.

Acknowledgment

Acknowledgment is due Dr. J. C. Wright for the use of his records and for his advice.

Discussion

Dr. J. T. Jennemann: The time is getting rather short, so I will confine my remarks to the treatment and touch on diet just a little bit. First of all, I want to compliment the essayist on his very valuable paper. I do not believe I could add anything to that. He presented it in nice form and the delivery was good.

Around St. Louis we do not get a great many of these cases. Last year I had five. We take a three percent solution of oxyborine and use that to clean up the mouth. Sometimes we use a solution of permanganate of potash, or

chlorate of potash, 2 drams, with about two drams of tincture of iron chlorid and water to make eight ounces. Use that as a mouth wash. We use trypacrin B solution intraperitoneally and to cause rapid absorption of this we use camphorated oil subcutaneously. I believe that will help the absorption quite a bit. We warm this before we inject it.

We also use gastro-intestinal lavage, using a bicarbonate solution. We first place our bicarbonate of soda in a small dish and pour boiling water over it so as to eliminate some of the gas. Sometimes, if you use it cold or lukewarm, you leave enough in the stomach or in the intestinal tract to produce a little bloat, so we scald the bicarbonate of soda and I believe we get just a little better results. We withhold water. We allow them to have a little cake of ice to lick on and sometimes put a little lemon-juice over the ice, just to quench the thirst. We withhold all food except goats' milk in small doses. Start out with a half-teaspoonful for a dose. I have yet to see the first dog vomit after getting goats' milk. Now goats' milk is splendid in the evaporated form. You can buy it in cans. A firm in Los Angeles puts up goats' milk in tin cans. I have used quite a bit of that in practice and like it very well. It takes 20 minutes to digest goats' milk, so you see you can get very prompt digestion and they usually retain it. I have not seen a dog vomit goats' milk. We take care of the diarrhea in the same manner as the essayist.

DR. O. M. NORTON: In the South we have been vitally interested in it for a great many years. Only in the past three years have we been able to have much success with it. Now, two symptoms that Doctor Rife did not mention are very important in diagnosis of the so-called sore mouth of dogs. Some owners who have had many cases are able to diagnose and bring them to the hospital before the symptoms of sore mouth show up. There are two symptoms that come before we have any lesions in the mouth or have the slobbering. One of these is a shifting lameness, sometimes in one leg and sometimes in another. It is not always present, but there is a symptom that is always present and that is soreness of the throat. Our idea of sore throat is that the trouble starts principally in the throat of the dog and, if we get those cases when we first notice the sore throat, they are easy to treat. In sore mouth, with just a little pressure on the throat, it will appear to be very sore.

Another thing. For a long time we thought that we did not have any temperature. A day or two before these dogs show much in the way of symptoms, they do have temperatures running as high as 104 or 105°F. That temperature, by the time the dog begins to slobber and the mouth breaks down, has disappeared and the temperature is often normal. Another thing we have found, that may occur just in that locality, is filaria in the heart in sore-mouth dogs. Practically 90 per cent of the dogs we lose there, on postmortem show filaria in the heart.

Now the treatment that we use has been acriflavine and proflavine. In the old treatment of permanganate of potash and different intestinal antiseptics, we found we were using too strong solutions. We were destroying too much mucous membrane. We find that, if we can get them in the first stages, by the constant use of the flavines in the mouth we can handle them. We do not believe in starving the dogs, we tried that for three years. We start with milk, small amounts of it, in the first stages and, if the dog does not want to eat, we give milk anyhow and as often as every half-hour, if we have them in the hospital. The worst trouble we have has been with the diarrhea that follows. We have trouble in checking that. If you check it, you kill the dog and, if you do not check it, you sometimes lose the dog.

In certain places in our country it is moist and low. We find that in these places, that go back in history twenty-five years, men have been unable to keep any good hunting dogs around for as much as a year. Now if it is due to feeding, if it is due to pellagra, it looks funny, that they have sore mouth. Every dog that is taken there has it. Lots of our dogs have strength enough or immunity or something to protect them. They have not been able to keep a dog there in the last twenty-five years without getting sore mouth. We have "re-cured" cases of sore mouth. We had a dog the other day that had sore mouth in 1924 and again last June and he had a case in April and apparently recovered. He had been out hunting since and came in there the other day with another case of sore mouth.

DR. C. P. CAUGHMAN: I would like to ask Dr. Norton if he does not sometimes have the bowel trouble as one of the first symptoms, the diarrhea, not necessarily the bloody diarrhea, but that characteristic diarrhea that you have only in cases of black tongue. I differ with him in this way and I agree with Dr. Rife in not feeding your dog for at least three days from the time you begin treatment, because I believe the stomach needs absolute rest.

DR. NORTON: You have diarrhea sometimes before you have mouth symptoms, but you never have it before the sore throat. The first symptom of black tongue is soreness of the throat, except the cases of stiffness or lameness. During the last five years Dr. Gates, of Clarksdale, my brother, another veterinarian and myself have averaged from three to fifteen cases a day during the whole summer long. During that time we have had, I will say, conservatively, about 1500 to 2000 cases of sore mouth. Friday, when we left there, we had sixteen cases of sore mouth or black tongue in our hospital.

DR. RIFE: Dr. Norton spoke about finding filaria. Do you associate that finding with the cause of the disease?

DR. NORTON: That is something I want to find out. These cases come from certain localities along the river. These dogs invariably die or by the time they get well of sore mouth they collapse.

DR. RIFE: We have filaria infestations in our country, but not so heavy. We find a few cases but not in conjunction with sore mouth or black tongue. I have examined the blood of these black-tongue dogs in the hope that I would find some, but I have never been able to associate the finding of filaria with the cause of the disease.

DR. NORTON: The only thing we have found in the blood was piroplasmosis We thought for a while there was some connection, but we think now they are two different diseases.

DR. RIFE: Probably the reason we never catch the cases with temperatures is because we never see them early enough.

DR. W. G. BROCK: In some experiments conducted at College Station, they have absolutely proven beyond a doubt that sore mouth in dogs is a dietetic trouble. They have reproduced this disease. They have said also that they could not transmit it by inoculation, transfusion of the blood, etc., and I think when you study this a little bit you will find out it is a dietetic trouble.

DR. J. C. MELVIN: I wish to ask Dr. Norton if cases with those throat lesions have any cough, as in distemper.

DR. NORTON: No cough. I would like to answer Dr. Brock about the diet. You cannot produce typical cases of black tongue with any diet that you can feed the dog. It is only a secondary consideration.

DR. BROCK: I can only quote the conversation from some of the men who have done the experimenting at College Station. Dr. Blackburn there is quite an authority on black tongue. He specifically stated to me, at one of the meetings, that he has been able to produce sore mouth by feeding, by taking out one of the vitamins that he figures is a necessity. In his experiments he has fed yeast and the dogs would never come down, but he did not know exactly why. That was his statement.

DR. NORTON: I tried the same thing and they did come down.

DR. MELVIN: As a suggestion, I wish to say that it would be well for veterinarians to get in touch with Dr. F. E. Patterson, of Auburn, Alabama. I wish to say that we have had only three mortalities from black tongue and we have been very successful in bringing our dogs through and very few of them in the hospital for a very few days at the most.

DR. FLYNN: We are deeply indebted, I might say doubly indebted, to Doctors Wright and Rife, because they are associated in practice, and they have given their time to prepare those interesting papers, coming here all the way from Atlanta, leaving their business, and giving us something that is really worth while. I want to extend to these gentlemen the appreciation of this section.

PERORAL ENDOSCOPY

By J. G. HORNING *and* A. J. MCKEE

Houston, Texas

In dog and cat practice, peroral endoscopy includes laryngoscopy, bronchoscopy, esophagoscopy and gastroscopy. The field for this work is greater than the majority of practitioners seem to think. The dog, being a mouth-breather, has more opportunities for the aspiration of foreign bodies into the air-passages, and inasmuch as he uses his mouth in play with various objects, there is actually a higher percentage of such aspirations into the air-passages, as well as the esophagus, than there is in the human. The dog and cat, being greedy feeders, "wolf" their food and hence the esophageal choke. In cats we have more cases of pharyngeal choke than in dogs, and more of these cases in kittens than in older cats.

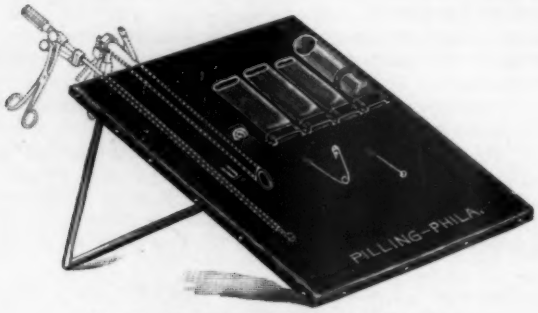

FIG. 1. Chevalier Jackson practice-board.

Owing the the nature of the anatomy of the dog, there is also a much higher percentage of voluntary expulsions in dogs than in human beings. In diagnosing foreign objects in the air- and food-passages, this can best be divided into three sections, namely, those involving the mouth, the larynx and the pharynx, all of which can be dealt with by the aid of the laryngoscope. Those affecting the trachea and bronchi can be handled by the aid of the bronchoscope, and those in the esophagus and stomach by the esophagoscope.

The greatest field for this work, as related in our previous articles, lies in the treatment of suppurative lung conditions, excessive amounts of tracheo-bronchial secretions, and other selected pathological conditions of the lungs, esophagus and

*Presented at the sixty-third annual meeting of the American Veterinary Medical Association, Lexington, Kentucky, August 17-20, 1926.

stomach. In diagnosing and treating these conditions, the X-ray plays an important part as a helpful adjunct to the endoscopy.

Instrumentation

The decided merit of this work in small animal practice has created deep interest among the majority of men specializing in this branch. However, the cost of complete instrumentation being prohibitive to the majority, after careful thought and consideration, we have devised a minimum working set that can be used on animals weighing from ten to thirty-five pounds,

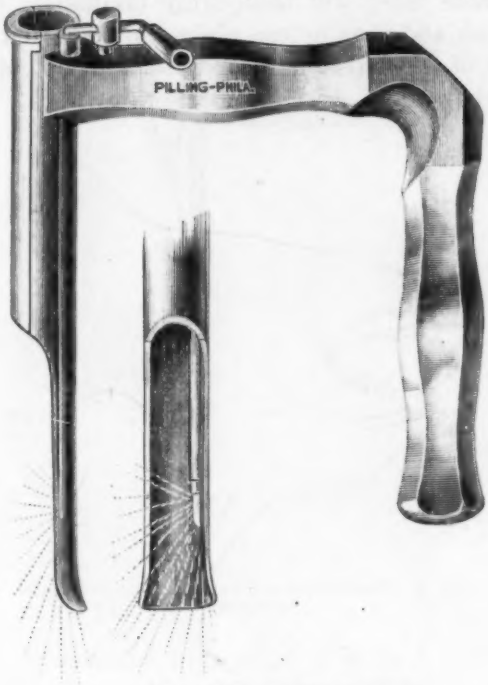

Fig. 2. Laryngoscope.

that will give the operator sufficient confidence in his ability and demonstrate to him the practical nature of this work.

As a foreword, it should be stated that when first buying such a set of instruments, he should start training the hand and eye by using them with the Chevalier Jackson practice-board, as illustrated in figure 1.

If one desires to study these parts carefully, with the landmarks, the first work can be carried out best on the cadaver. After becoming thoroughly familiar with the parts, practice the

removal of foreign bodies placed at various points in the bronchi. In aspiration of the trachea, for the removal of bronchial secretions and pus, for practice on the cadaver, milk-and-egg solution can be injected to represent tracheo-bronchial secretions or pus and thus get the absolute modus operandi for use on the live subject.

After sufficient practice on the cadaver to give confidence in distance, get a dog from the pound and narcotize him with morphin sulphate, by subcutaneous injection of one-sixth of a grain for each pound of body weight. Practice on this subject, along the same lines, will thoroughly familiarize the operator with this work and then he can make the operation on the paid patient that of the experienced operator and not of the novice, and insure the patient receiving meritorious work.

FIG. 3. Placement of cork on canine teeth to hold the mouth open

For cats, use 1 cc of a 2 per cent solution of chloralose to every pound of body weight. After the patient is narcotized, the laryngoscope shown in figure 2 can be inserted into the mouth, the patient lying in posterior anterior position, the lip of the laryngoscope being gently traced along the tongue, back into the larynx and the landmarks and anatomical parts studied carefully. This will give the operator an idea for mouth and larynx work. In the dog the mouth can be held open with a Gray mouth speculum and in the cat with a cork, as shown in figure 3.

The irrigating and aspirating bronchoscope, shown in figure 4, can be inserted into the mouth and, at the chink of the glottis,

inserted into the trachea. The trachea can be examined by moving the scope from side to side and, by slow and careful examination in this manner, one will be able to see when he comes to the carina or bifurcation of the right and left main bronchus.

If the examination is not carried out carefully, one will go directly from the trachea into the right main bronchus. It is

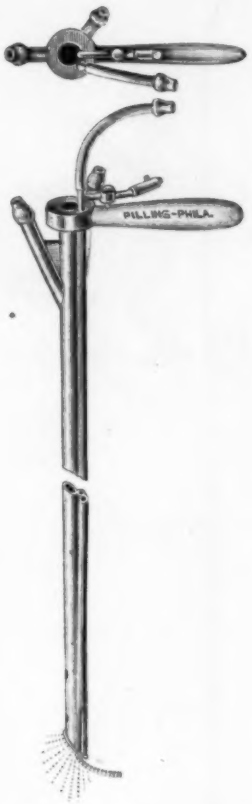

FIG. 4. Irrigating and aspirating bronchoscope.

always well to bear in mind that careful study has to be given to the anatomical parts. The absolute knowledge of the various landmarks is essential, otherwise thorough bronchoscopy would be an absolute farce.

Bronchoscopic work can be used for the removal of foreign bodies and for the diagnosis and treatment of lung conditions. In the aspiration of tracheo-bronchial secretions and suppurative conditions of the lung, we use a curved aspirating-tube, shown

in figure 5. In the treatment of suppurative conditions of the lungs and also in asthmatic conditions, we use a Clerf atomizer, shown in figure 6.

In the Clerf atomizer, we use a 2 per cent gomenol solution to spray the parts after aspiration and, in asthmatic conditions,

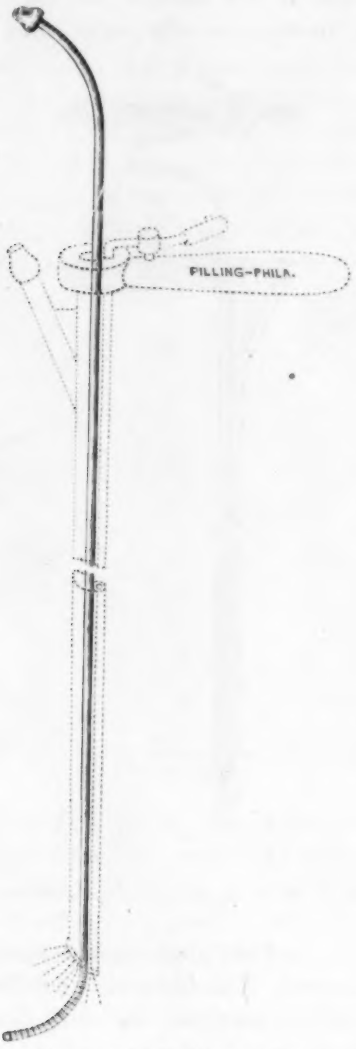

FIG. 5. Curved aspirating tube.

we aspirate the secretions and spray with a solution of epinephrin and butyn (Abbott). This is also used in diseased conditions of the larynx. We use the epinephrin and butyn solution to spray

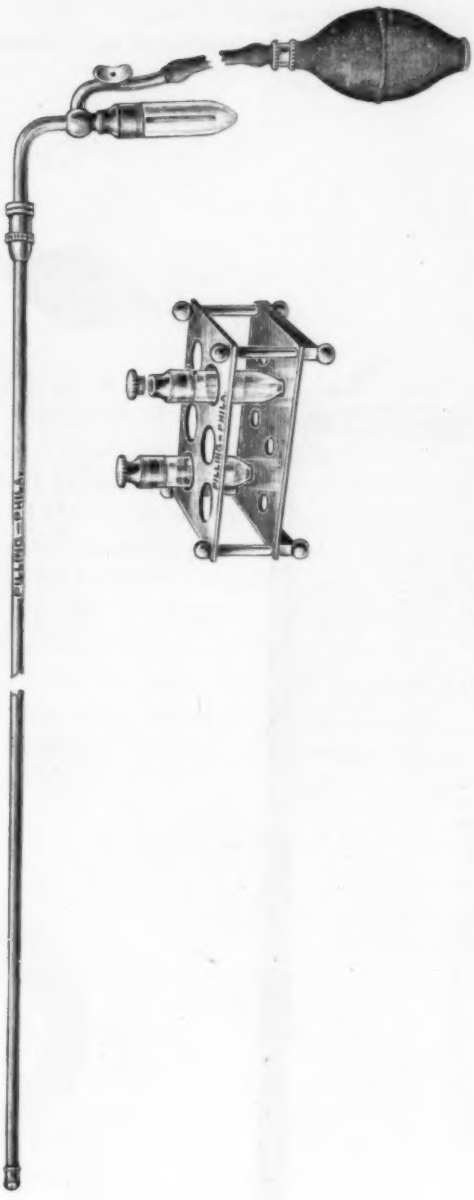

FIG. 6. Clerf atomizer.

the tonsils in tonsillectomy. The laryngoscope described is of great value in illumination of the field for this work.

In esophagoscopic and gastroscopic work, one will find the ballooning esophagoscope and gastroscope, shown in figure 7, of great value. This will do in treatment of diseased conditions

FIG. 7. Ballooning esophagoscope and gastroscope.

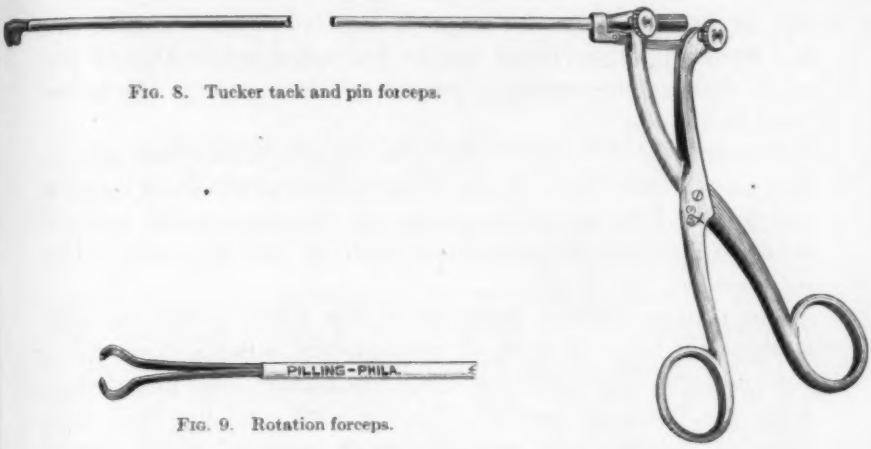

FIG. 8. Tucker tack and pin forceps.

FIG. 9. Rotation forceps.

of the esophagus, as in inflammation due to swallowing of lye, swallowing of foreign bodies, and in the treatment of gastric ulcers. The cases of inflamed mouth and esophagus from lye, that we have had to treat, have occurred in instances where the client had lye on the floor, or was cleaning some material with lye, and the dog ran over the solution. As soon as it commenced to burn his feet, he licked them and produced severe inflammation of the mouth and esophagus. Direct surface treatment is indicated in chronic gastritis.

In full examination in this work, the scope is used with the window-plug, permitting sighting of the tissue area, and by pumping and placing sufficient air-pressure ahead, the parts

FIG. 10. Battery for scopes.

FIG. 11. Battery cords.

can be ballooned as the scope is inserted. On entrance into the stomach, the stomach can be ballooned sufficiently to permit a thorough inspection of its entire surface, except the uppermost cardiac portion.

For removal of foreign bodies, we use the Tucker tack and pin forceps, shown in figure 8, the rotation forceps shown in figure 9, and for the lighting of our scopes, we use the practice battery, shown in figure 10, in conjunction with the battery cords, shown in figure 11.

The instrumentation described in this article is the minimum working set for good work on medium-size animals presented in this specialty. They will cost about one hundred and fifty dollars. After one becomes familiar with the work and has had many hours of practice and sees the many cases in which peroral endoscopy may be used in daily hospital work, he can then add further instrumentation to his equipment.

PROMINENT VETERINARIANS IN BAD ACCIDENT

While returning to Bloomington from Springfield, where they had attended a hearing before the Agricultural Committee of the Illinois Legislature, Dr. T. A. Sigler, president of the A. V. M. A., Dr. W. H. Welch, ex-president of the A. V. M. A. and secretary of the Illinois State Veterinary Medical Association, and Dr. J. S. Koen had narrow escapes from death in an automobile accident, Wednesday evening, May 18.

The accident happened near Shirley, after the party had left Williamsville, in a driving rain. Dr. Koen was driving his coupe, with Drs. Sigler and Welch beside him. A moving van was parked in the middle of the pavement, with only one white light showing, at the rear. The occupants of the car thought the van to be another car approaching them. Dr. Koen turned to the right of the road as far as possible before he realized what was ahead. In attempting to turn again, for the purpose of passing on the left, his car skidded into the rear of the van. Fortunately it was an all-steel body, or all three might have been instantly killed.

Dr. Sigler received a bad cut on his right hand and it is believed his sternum is fractured. Dr. Welch received fractures of one collar-bone and two or three ribs. Dr. Koen's arm was badly cut and his left side severely bruised. The car was completely wrecked.

THE ENDOCRINE GLANDS WITH REFERENCE TO VETERINARY MEDICINE*

By HERMAN J. ACHARD, *Glendale, California*
Managing Editor, The Endocrine Survey

In the JOURNAL of the American Veterinary Medical Association for March, 1923, Dr. J. F. Shigley discussed the glandular-group antagonism and its application to veterinary practice. In this very interesting paper, a table is given showing the periods of glandular activity and the important endocrine glands related to each of the different age-periods. You will find there, in the first age-period (youth), the following glands active: thymus, pituitary, thyroid, adrenals, pineal. In the second period, or that of sexual maturity, there are (for the male): testes, adrenals, thyroid, pituitary and prostate; (for the female): ovaries, adrenals, thyroid and pituitary. During pregnancy, the placenta, corpus luteum, mammary glands, fetus and uterine mucosa are active. While the mother is suckling her young, the mammary glands, ovaries and uterine mucosa play an active part. In the last age-period, or that of old age, all the glands of internal secretion atrophy.

PITUITARY

As far as we can tell, the thyroid and the anterior pituitary are responsible for growth and development. In its normal functioning, internal secretion of the anterior pituitary stimulates the growth of the youthful organism—"according to Hoyle," as it were. An excessive functioning of this endocrine gland is responsible for what we know as acromegaly, which is observed in animals as well as in humans. An insufficient functioning of the anterior lobe of the pituitary will leave the organism in an infantile condition, with retarded development, both physical and mental.

The posterior lobe of the pituitary produces an internal secretion that maintains the tonus of the capillaries and assures the contractile power of the smaller vessels. It stimulates the smooth muscular fibers, causes vaso-constriction, and increases blood-pressure by its direct action upon the blood-vessels, while the pressor effect of adrenalin is exerted upon the nerve-endings.

*Read before the Southern Califorina Veterinary Medical Association, Los Angeles, California, December, 1926.

The pituitary internal secretion, through its effect upon the smooth muscular fibers, influences peristalsis, and is used in the human to overcome post-operative intestinal paralysis. Pituitrin is used in inertia and is often of service in terminating tardy labor.

Thyroid

The thyroid, as well as the pituitary, aids in regulating growth and more especially in stimulating metabolism. It seems to be the key, the switch, as it were, that starts and maintains the metabolic processes. If the thyroid is removed, sparing the parathyroids so that life is preserved, all vital processes are promptly slowed up. A sluggish metabolism occurs, frequently associated with constipation. There is not enough heat in the body, which is constantly chilly, cold. The heart-beat is apt to be slow, the temperature below normal, the respirations are liable to be slowed down, and the mental processes to be sluggish. Because of insufficient utilization of the food particles, there occurs an infiltration of the skin and of the subcutaneous tissues, the infiltrating substance being mucin. The skin is poorly nourished and, therefore, is dry, exfoliating, and harsh. With the failing detoxication, the blood-pressure frequently rises, and altogether the organic processes are retarded and unsatisfactory.

On the other hand, if the thyroid works overtime, thus producing an excess of its internal secretion, the metabolic processes are hastened. Food is broken down and subjected to combustion at an increased rate, and a good deal of it is lost. Diarrhea is frequent. Because of the increased rate of oxidation, the labile temperature is very liable to go high, as does the pulse-rate—in fact, tachycardia is characteristic of hyperthyroidism—and great nervousness and irritability ensue. Often the eyeballs protrude, exophthalmus being a characteristic of hyperthyroidism. Tremor, which also shows the irritation of the nervous system, is another symptom.

There is such a thing as qualitative disturbance of the thyroid internal secretion, which is different from seeming hypothyroidism or hyperthyroidism, and amounts to a dyscrinism or a perversion of function. This, I think, is at the bottom of toxic thyroid adenoma, which is an extremely serious disease and not nearly so susceptible to treatment as is either myxedema or exophthalmic goiter. In fact, it usually calls for surgical removal.

Parathyroids

It was in the eighties of the last century that Professor Kocher, of Bern, Switzerland, first amputated thyroid glands which had degenerated into goiter, and observed a cachexia strumipriva and very rapid degeneration of the entire organism which promptly led to death. In time it was found that this sequel of the operation was not attributable to the total extirpation of the thyroid but to the loss of the parathyroid glands, four tiny glandular bodies situated at the four corners of the thyroid, sometimes deeply embedded in it. In man, they are about the size of a lentil, or even smaller; they are of different histological structure from the thyroid, and can easily be distinguished by the operator. They were recognized as independent organs, in 1892, by Sandstrom, of Stockholm. Unfortunately, Shigley omitted mentioning the parathyroids in his list of essential glands, for they are just as vital as are some of the other glands —the adrenals and the anterior pituitary, for instance.

Removal of the parathyroids causes, first of all, an irritability of the nervous system which manifests itself in tetanoid convulsions and which may lead to death by way of a rapidly progressive cachexia. This nervous upset is due to the fact that the blood has lost its calcium, through which the nerve balance is evidently maintained, and it has been shown that the chief function of the parathyroids is the maintenance of a normal calcium metabolism. By this I mean that a proper content of fixed lime is maintained in the blood, which should amount to 0.9 mg. per 100 cc.

Adrenals

The adrenals are the depository where the organism keeps its supply of pep. A lack of the adrenal internal secretion produces weakness, debility, cardiovascular imbalance, a lowering of the blood-pressure, digestive upset, emaciation and, ultimately, death. The vigor of the circulation and the blood-pressure are maintained by the internal secretion of the adrenal medulla which produces adrenin, or epinephrine, which is active in a tremendously high dilution. I think I have read somewhere that as little as one-millionth of a milligram exerts a definite physiological effect. Epinephrine contains the pressor principle by which the blood is pushed through the arterial system with sufficient force, and it is supplemented in this pressor effect by the internal secretion of the posterior pituitary.

The adrenal cortex has an antitoxic function, and it also seems to have some association with the gonad functions. At any rate, an excessive action of the adrenal cortex causes precocious sex development. In women, it gives rise to virilism, through which male characteristics declare themselves, such as a deep, base voice, growth of beard, disappearance of feminine fat distribution, male conformation of the body, etc. It has been suggested that nymphomania, which is frequently a serious problem with animal fanciers, has an adrenal origin. It is almost always caused by tumors of the adrenal cortex, and is associated with ovarian atrophy.

The chapter on blood-pressure is rather an interesting one, especially in human medicine, because there it comes to our consciousness more. I assume that in animal practice also, it produces considerable interest. As already said, a normal blood-pressure is maintained by a proper functioning of the adrenal medulla and the posterior pituitary. Any excessive action on their part is held in check by the internal secretion of the liver, so that both pressor and depressor functions are maintained in balance. If, through any cause, the adrenal internal secretion is irritated, as happens through toxins, it may get the best of the balancing depressor action of the liver; or, if the liver's internal secretion is falling down and cannot manage a sufficient degree of detoxication, the adrenal pressor effect will run away and a high blood-pressure will result.

Thymus

The thymus has frequently been denied inclusion among the endocrine glands, yet the opinions advanced to justify its inclusion are just as important as are those that would tend to exclude it. This gland is essentially one of childhood, yet, occasionally, it is encountered in adolescents and adults. Although the functions of the thymus are not fully recognized, it seemingly has to do with the reorganizing of lymphoid structures. It certainly is associated with the gonads, as is shown definitely by its extirpation. In young animals in which the thymus has been removed, there occurs a dimunition in stature, and also in the volume of the bones. The latter lose their lime, become flexible, and often (as in osteomalacia) the general nutrition is retarded. Growth is slow, and the testicles and ovaries are arrested in their development. In the course of time, young animals deprived of their thymus glands become somnolent, apathetic and idiotic.

In castrated subjects, the thymus is larger than in control animals of the same litter that have not been deprived of their gonads. Some authors have recorded that in exophthalmic goiter the thymus persists or is hypertrophied.

These are various observations that have been adduced in support of the idea that the thymus is an endocrine gland.

Hypertrophy of the thymus manifests itself in dilatation of the veins of the face and neck. There is a permanent or intermittent dyspnea and attacks of suffocation and heaving. These symptoms are exaggerated when the head is extended much, and are explained by the fact that there is a compression of the trachea, the veins and the nerves at the opening of the chest (pressure on the pneumogastric). Thymic hypertrophy may be associated with status thymico-lymphaticus, which is characterized by increase in the thymus and the spleen, by hypertrophy of the lymphadenoid tissue throughout the body, and by hypoplasia of the adrenal system.

From the endocrine viewpoint, the thymus seems to be associated with the gonads in such a way as to retard or to prevent their premature development. A similar function has been attributed to the internal secretion of the pineal gland. It is certain that in cases of sexual precocity in children, either boys or girls and as young as three years of age, tumors of the pineal gland or atrophy of the thymus have been found. In other words, either one or both of these glandular organs were incapable of functioning. Another reason for relating this balancing function, or antagonistic function, to that of the gonads is, that during adolescence, when the gonad function is becoming established, both the thymus and the pineal gland undergo, normally, a process of involution.

Gonads

From a general viewpoint the gonads are most important because of their external secretions, namely, the semen, in the case of the males, and the ova, in the case of the females—both of which have to do with the reproduction of the species. The maintenance of these external secretions is largely under the influence of the internal secretions which the gonads produce. The internal secretions of the testes and of the ovaries are also concerned in the regulation of growth and in the maintenance of the mineral metabolism. The former determines the male-

ness of the organism, whether animal or human, and the latter determines the femininity of the organism.

Of particular and definite importance to us as practitioners of medicine, whether for animals or for humans, is the functioning ability of adult glands with regard to reproduction and to the various problems associated with sex. Limiting ourselves to veterinary medicine, the ability of the male animal to serve the female in heat and to fertilize her, is of interest; and the readiness of the female animal to accept this service and to bring forth a litter of healthy young, is likewise of importance. Frigidity and sterility usually are associated with some irregularity in the gonads and are frequently subject to correction by suitable endocrine therapy. It is quite proper that Shigley has based his recommendations for hormone therapy upon the fact that not one of the endocrine glands can function without any reference to the others, but that they are all associated in one republic, so to speak, and that there is an interrelation of glandular functioning which determines normal processes. For that reason, such conditions as frigidity or sterility in animals are frequently corrected by a suitable pluriglandular therapy combining ovarian, pituitary, thyroid, and adrenal substances. Sterility in the male, which is not mechanical—that is to say, is not due to deformity of the organs—may be corrected, in many cases, by a corresponding pluriglandular treatment that includes the testicular and other endocrine substances.

In human medicine, pregnancy is frequently associated with intoxications leading to serious disturbances. We have, for instance, vomiting of pregnancy; later, we have nervous upsets which may become so serious as to amount to uremic convulsions, eclampsia, etc. I do not know how serious this factor may become in veterinary medicine, but probably it is active. It may be said that the toxemia of pregnancy leading to vomiting is frequently controlled by a combination of placental substance, thyroid and lime. In cases in which eclamptic convulsions threaten, it is believed that the detoxicating functions of the liver and the thyroid are in abeyance, and for that reason the condition is properly treated by feeding desiccations of thyroid and hepatic parenchyma. It may be treated by an hepatic extract which has a very prompt and stimulating effect upon the detoxicating function of the liver and, incidentally, may lower the alarmingly high blood-pressure. This effect of hepatic extract was discovered about a year ago, by Dr. Mac-

donald, of St. Catherine's, Ontario, Canada, and has been studied in several cities in this country and abroad—incidentally, in our own city, by Dr. Henry R. Harrower.

The onset of labor seems to be distinctly an endocrine process. During pregnancy, the occurrence of heat in female animals, or the menstrual flow in women, is prevented by the corpus luteum of pregnancy, which checks the ovarian endocrine function. At the end of pregnancy, the corpus luteum undergoes a process of involution, and for this reason its restraining influence upon the ovarian function proper is lessened. The ovarian function through which uterine contractions are produced is intermediated by the posterior pituitary. In other words, the ovarian internal secretion stimulates uterine contractions by way of the posterior pituitary, and it has already been mentioned that the posterior pituitary internal secretion has a similar effect upon the adrenal muscular fibers. When the restraint exerted by the corpus luteum is removed, the stimulating effect exerted upon the uterine muscles by the ovarian and the posterior pituitary internal secretions is able to manifest itself, and uterine contractions are no longer checked but become active, and the uterus discharges its contents. If, for any reason, labor is tardy, there is a time in its course when it is not only safe but wise to administer a preparation of the posterior pituitary in order to encourage uterine contractions in the expulsion of the fetus. Of course, it should not be given if there is a mechanical obstruction in the shape of a deformed pelvis.

After labor, the promptness with which the uterus contracts, and, in doing so, closes the existing sinuses, determines the degree of hemorrhage that takes place. Clinical observation has shown that, when the young commences to suckle, the uterus contracts, vigorously expels clots of blood that it may contain, and ceases to bleed. This has suggested the conclusion that the mammary gland produces some substance that checks hemorrhage from the uterus. Clinical observation also has confirmed this conclusion, and the administration of desiccated mammary substance is actually used with success, either alone or in combination with posterior-pituitary substance, in checking excessive menstrual bleeding, especially during the climacteric.

One important point after labor is the ability on the part of the mother to suckle her young. With regard to this, actual observation again has suggested a reason for the prompt production of milk. It is well known that domestic animals, notably

cows and cats, eat the placenta, and it has been concluded that this is not merely for the purpose of removing an offending substance, but that there is a definite reason for it. As a matter of fact, the ingestion of placental substance after confinement stimulates the production of milk. This has been verified not only in human medicine but in veterinary practice also.

Therapy

Time permits only a few remarks on endocrine therapy, which presents a wide field of possibilities. It is important, of course, to obtain reliable preparations, if one is to expect results. Empirical therapy has enriched our materia medica considerably, as it has done so often in the past.

Adrenal support has proved very beneficial after infectious diseases, for instance, distemper.

Parathyroid is helpful in diseases of metabolism, in rickets, osteomalacia, in slow-healing ulcers, also in arteriosclerosis, gout, hives—in short, in any condition in which there is a deficiency of lime. If care is not given to proper feeding, pregnancy may constitute a calciprivic condition, as may also tuberculosis.

When parathyroid is prescribed, care should be taken to supply lime at the same time. The diet also must be well rounded out and must contain all the necessary vitamins in addition to the calorie-bearing substances.

A very interesting study, concerning the calcium requirements of the human and animal organisms, was published a few years ago by Prof. Oscar Loew, of the University of Munich, Germany. Those who read German will find this text of tremendous interest. In fact, I have been so impressed with it that I should like to translate it into English, if only I had the opportunity. Dr, Loew points out that animals that do not get enough lime often do not conceive or else they miscarry. If they start giving milk, they go dry very soon. Frequently their bones soften and become deformed (osteomalacia). He urges the deliberate administration of chlorid of calcium as best calculated to supply the requirements of the animal organism, and insists that neither calcium carbonate nor calcium phosphate give satisfactory results.

Thyroid may be called for at some period during pregnancy, also in chronic rheumatism, in many of the dermatoses, and in obesity. It may be associated with pituitary or ovary, or with both.

Placenta substance has proved clinically useful in establishing an immunity to placental protein poisons, and thus in overcoming the nausea and vomiting of pregnancy. It is of great value also as a galactagogue and uterine involutant.

CORN INSTEAD OF GASOLINE

Veterinarians in agricultural districts have frequent opportunities to discuss the horse-vs-motor question with their clients. At this time, when farmers are wondering what they are going to do with their surplus crops, there are but few of them who ever stop to analyze the situation, few of them who are aware that they are almost entirely responsible for their dilemma, and few who realize that they hold the solution of the problem in their own hands.

Dean Curtiss, of Iowa State College, has suggested a way to cut down the surplus, or, more accurately, to restore something like a balance between production and consumption. Most, if not all, veterinarians will concur in the conclusions drawn by Dean Curtiss in his analysis. The facts and suggestions are worth passing on. They are as follows:

The farmer's chief trouble is obviously a surplus of corn, and consequent depression of prices. The remedy lies in smaller production or increased consumption. The present trouble is due to declining consumption, and the farmer himself is to blame for the situation, to the extent that he has substituted mechanical power produced by gasoline for horse power produced by farm feeds.

The horse will not again occupy his former place in city dray work, nor will he ever again be restored to the service usurped by the automobile.

The farm motive power is different. More motive power is used on the farms of the United States than in any other industry. Horse power is, in the last analysis, the economic power on the farm.

There are now about 5,000,000 less horses in the United States than there were ten years ago. That number of horses would consume more than the annual surplus of corn and oats. If motorization of the farms goes on, the surplus of grain will increase. If the horse-breeding industry is not restored, motorization of the farms will be imperative. The farmer himself holds the key to the situation. If he chooses to substitute gasoline for corn, he will, in the end, pay more for gasoline and take less for corn. The farmer who raises corn to sell will pay the penalty. The stock farmer will fare better, though cheap corn tends to produce a surplus of livestock products.

The farmer who substitutes gasoline for corn in producing motive power should weigh well some fundamental facts:

1. Gasoline-driven substitutes for the horse do not consume any farm products. On the contrary, they entail heavy expenditure for initial purchase and operating cost.
2. They do not reproduce themselves on the farm, but entail heavy expenditure for renewal.
3. They leave no fertility on the farm, but the practice growing out of their use tends to impoverish the soil.
4. They displace an important outlet for farm grains.

BANG BACILLUS DISEASE (BOVINE INFECTIOUS ABORTION)*

Prevention, Control and Eradication in Pennsylvania

By T. E. MUNCE, *Director*

Pennsylvania Bureau of Animal Industry, Harrisburg, Pa.

The state of Illinois is so situated geographically that a well-organized and administered live stock disease prevention and control organization is of the utmost importance to the health of live stock of almost every other state in the Union. I say this for the reason that within the borders of the state of Illinois are situated the great Chicago, also the East St. Louis stock-yards, from which live stock in vast numbers are distributed to other sections of the United States.

Besides being one of the largest live stock-distributing states in the Union, the state of Illinois is among the greatest agricultural and live stock-producing states, which makes the prevention and control of animal diseases of great economic importance to the people within the State. Illinois ranks third among the states in value of farm crops; second in value of farming land and buildings; second in animal products; second in number and value of swine; second in number and value of horses; eighth in number of cattle; sixth in value of cattle, fifth in number of milch cows and fifth in value of milch cows. This ranking places your state among the most important of agricultural and live stock-producing states.

The United States Department of Agriculture, in its 1925 Year Book, estimates the value of the live stock in Illinois as follows:

	Value
Cattle and calves	$118,950,000
Swine	76,412,000
Sheep	8,303,000
Horses	71,905,000
Mules	14,380,000
Poultry	25,235,000
	$315,185,000

*Presented at the annual University Veterinary Conference, University of Illinois, Urbana, Ill., Feb. 16, 1927.

It can be observed by referring to the above data that cattle alone have a value of nearly one-half the total value of all live stock in the state of Illinois, and considerably more than one-third of the value of live stock and poultry combined. The prevention and control of cattle diseases, therefore, occupy a most important position in the work of an organization constituted for the study, prevention and control of live stock diseases in your state.

For the live stock industry of your state and all other states to prosper and function at its highest rate of efficiency it must be protected from disease. President Coolidge has well said, "The key to successful agriculture is its herds and flocks." Therefore, I say that healthy live stock are essential to a prosperous agriculture.

Success in the business of breeding and raising cattle for dairy and beef purposes or the business of maintaining cattle for dairy purposes only is dependent upon many factors, the most important of which is *health* of the individual members of the herd. In addition to being essential to the production of wholesome milk and beef, health is also essential to high efficiency in both breeding and productive dairying. Prevention of diseases is indispensable to a high rate of herd efficiency and is the *key* to a successful breeding or dairy business.

The basic principles for the prevention, control and eradication of all transmissible diseases are practically the same. The application of the principles of sanitation including disinfection in an attempt to prevent and control one disease cannot help but aid in the prevention and control of other diseases. The best-known methods for the prevention and control of all transmissible diseases have basic principles of sanitation. All transmissible diseases have a specific cause, which is more or less different for each individual disease. Most transmissible diseases can be prevented and it is best to do so. The cause of a disease cannot be present with susceptible animals without the presence of disease. Diseases are best eradicated by removing the causative agent. On the other hand transmissible diseases are controlled by limiting the action of the cause or by removing the susceptible animals. In all cases sanitation plays an important part.

Symptoms and Diagnosis

The act of aborting is probably the most important clinical manifestation, at least from the standpoint of the amount of

damage done to surrounding members of the herd. Visible clinical symptoms other than the act of aborting are seldom observed. The average herd-owner does not have the proper conception of the term "contagious abortion," which is the term under which he knows the disease caused by *Bacillus abortus* (Bang). He considers any premature delivery of a calf as abortion and is very prone to consider it accidental. If he does consider these abortions frequent enough in occurrence to convince him that the trouble is contagious, he usually does not understand that there is any more to the disease than the manifestation of this symptom.

The sooner and more thoroughly both veterinarians and herd-owners understand the fact that Bang bacillus disease is a specific transmissible disease having a specific cause which is readily transmitted from a diseased to a healthy animal, the more general and successful will be the attempts to prevent and control this preventable and wasteful disease.

In arriving at a diagnosis from a clinical standpoint, the history is very important. Bang bacillus disease in a herd is usually traceable to the addition of infected cattle to the herd, or exposure to infected animals in some other way. Any agency which serves as a means of introducing *Bacillus abortus* (Bang) in live form to susceptible animals may be considered a source of infection. Therefore, in the prevention and control, one's aim should be to keep the cause and susceptible animals apart.

While Bang bacillus disease can usually be diagnosed with a considerable degree of accuracy from a clinical standpoint, through the observance of the herd manifestations, much time is wasted in waiting for these manifestations to be presented and much damage has often been done which could have been prevented had more conclusive methods of diagnosis been used.

The only positive proof of the presence of Bang bacillus disease in a herd is obtained by finding the Bang bacillus, or by applying specific tests to the blood from animals of the herd.

Importance

Probably no other disease of cattle is at the present time receiving so much serious thought of the wide-awake breeder and dairyman as is Bang bacillus disease. For, as pointed out, no other disease now prevalent in this country is deserving of as much thought from the standpoint of herd efficiency. Moreover, no other prevalent disease handicaps so much the improve-

ment of breeds of cattle or the economical production of milk and beef.

Bang bacillus disease strikes at the heart of the cattle-breeding and dairy business, destroys the very nucleus—the calf, and thus defeats the purpose of the breeder. Moreover, the efficiency of herds infected with the bacillus of Bang is much reduced both from breeding and milk-production standpoints.

Cow-testing associations enable dairymen to determine which cows are profitably maintained and which cows are maintained at a loss. How often in selecting cows by this method do owners or herdsmen take health into consideration? Dairy and animal husbandrymen, in their lectures on profitable dairying and breeding, should emphasize the importance of health more than they are now doing.

The veterinary profession has preached for years that attention to health and sanitation is indispensable to successful live stock husbandry.

The official tester in a prominent cow-testing association in Pennsylvania has pointed out that reduced efficiency in cows in his district is due mainly to disease—bovine abortion and tuberculosis; that 65 per cent of the cows are sent to the butcher on account of their failure to reproduce because of bovine abortion.

A prominent Pennsylvania dairyman and breeder stated that with what is considered a fair price for milk, dairymen cannot afford to keep cattle and bovine infectious abortion in the same barn. He stated further that the chief complainers of the price obtained for milk were those whose herds were polluted with disease; that they could make a profit at the price then current by reducing the cost of production through the elimination of preventable disease, thus causing increased production.

The average dairyman frequently places little or no commercial value in calves, yet in order for his business to be successful he must have high milk-production. In order to get this needed production the cow must be in a normal state of health and must perform her normal physiological function approximately once a year by giving normal birth to a normal-time calf, else the normal stimulation of milk-production is not brought about. The breeder who is not interested in dairying, in order to produce high-valued animals, both calves and milk-production, must have both calves to be grown to maturity and must have these calves backed by high records. The beef-producer is dependent upon calves to be grown to maturity for the production of beef. None

of these ends can be successfully attained if the cows abort their calves. What then is more necessary to the dairy and beef industries than the maintaining of healthy animals? What is more essential than the prevention, control and elimination of disease? Provide sanitary quarters for the healthy calf and subsequently for the cow and this end will have been accomplished.

Prevention and Control

Ever since the discovery of the specific cause, the bacillus of Bang, thirty years ago, the Pennsylvania Bureau of Animal Industry has tried different methods of control. The greatest amount of success was obtained through the practice of principles based on sanitation. Today, the most successful method known for the prevention and eradication of Bang bacillus disease is based on the principles of sanitation, in addition to which the abortion blood-test is used to determine the presence of infection in individual members of the herd and as a guide in handling the entire herd.

The blood-test is used to determine which animals are infected with Bang bacillus disease, without regard to visible symptoms. A positive reaction to the blood-test indicates that the animal is infected with Bang bacillus disease. A reaction does not indicate that the infected animal ever has or ever will show the characteristic symptom by aborting, any more than a positive reaction to the tuberculin test indicates that the reacting animal ever has or ever will show physical symptoms of tuberculosis.

The study of methods of prevention and control of Bang bacillus disease, up to the time a definite plan was adopted, constituted an eliminative study and the methods applied were apparent and not actual.

Actual control is a means of determination and elimination of disease and diseased animals and the practice of recognized methods necessary for the safeguarding of the herd.

Prevention measures, practiced in conjunction with sanitary principles, constitute the best-known and most profitable method of handling Bang bacillus disease.

About seven years ago a special study of Bang bacillus disease was begun by the Pennsylvania Bureau of Animal Industry, and while eliminative studies were continued, definite principles based on blood-testing, elimination of reactors and sanitation were placed in operation in a few Pennsylvania herds. After these principles were practiced over a sufficient period to justify

the conclusion that satisfactory results in the control of Bang bacillus disease were being obtained, the first Pennsylvania Plan for the prevention and eradication of Bang bacillus disease was drafted. It contained the principles which had been in operation in these few herds from one to two years, and in operation in one herd where infection had been present to a small extent for a number of years. The first "Abortion-Free" certificate was issued, December 16, 1921.

Pennsylvania Plan

When the Pennsylvania Plan was first proposed it met with objections and opposition, as is usually the case with all new things. Many breeders, and indeed some veterinarians, thought the plan neither feasible nor necessary and suggested that not enough was known about the disease to justify our attempting to prevent and eradicate it and the time was not ripe for launching our plan for that purpose.

The objectors reminded us of an ancient verse and Persian proverb as follows:

"He who learns and learns yet does not what he knows
Is one who plows and plows yet never sows."

Some of us believed differently, however, and in the face of existing opposition and discouragement the Pennsylvania Plan was put forth and made available in 1920 for those who desired to take advantage of it.

At the time of the adoption of definite constructive principles for the prevention and control of Bang bacillus disease, breeders and dairymen were pressing our state disease-control organization for definite advice which would bring forth lasting results. We believed that the adopting of such a standard would benefit the cattle industry in many ways:

1. Improve the quality of the herd. Increase the quantity and quality, and decrease the cost of production.

2. Establish a definite means of prevention and control for those who desired it.

3. Encourage stockmen to establish abortion-free and more highly efficient herds.

4. Provide a safe place for prospective purchasers to obtain new animals.

5. Educate dairymen and breeders to the necessity of veterinary supervision.

6. Make all producers of live stock and milk maintain in their management a higher degree of sanitation.

7. Create more public interest in dairy products.

The Pennsylvania Plan for the Prevention, Repression and Eradication of Bang Bacillus Disease

(Bovine Infectious Abortion)

Made Dec. 1, 1921; revised Dec. 1, 1924, and Dec. 1, 1925

1. The Pennsylvania Plan for prevention, repression and eradication of Bang bacillus disease (Bovine Infectious Abortion) is a plan based on sanitary principles and includes repeated blood-testing of the herd and elimination of reactors.

A single blood-test will not disclose all animals infected, for the reason that in some animals the disease may be in the incubative stage at the time of the blood-test. Unsuccessful attempts have been made to eradicate Bang bacillus disease from herds where the reactors were separated in a separate row in the same barn and where reactors and non-reactors went into separate pastures. This method approaches the plan and could be carried out in some herds wherein it does not seem advantageous to carry out all details of the plan, provided the reactors are not re-bred and are disposed of as soon as the amount of milk produced is reduced to the point where it is unprofitable.

In herds where there is a high percentage of reactors and the adoption of the plan in its entirety does not seem practicable, one should approach it as nearly as possible in preparation for its full adoption later. The calves should be raised separately and kept free from disease to replace the reactors in the mature herd later. They should pass at least two negative tests before being bred.

2. An abortion-free, approved herd, under the Pennsylvania Plan, is one in which no evidence of Bang bacillus disease has been shown and in which no reactors to the abortion blood-test have been found, covering a period of at least one year.

A herd is required to be negative to blood-test (free) over a period of at least one year, for the reason that some animals do not react their first time until after having calved normally or after having aborted. For a herd to have been negative to repeated tests for a period of one year gives (practically) all cows in the herd time to have calved and to have been blood-tested after having calved.

3. To benefit by the plan, the owner must place his herd under supervision of the Bureau of Animal Industry, Pennsylvania Department of Agriculture, for the prevention, repression and eradication of Bang bacillus disease.

The law makes it the duty of the Bureau to supervise the control of transmissible animal diseases. All disease control work should be conducted after a definite plan, tending towards uniformity.

4. No herd will be placed under supervision, the owner of which is not practicing measures against tuberculosis.

Tuberculosis eradication is a public-health measure and should have preference from that standpoint. Abortion control benefits the herd-owner from an economic standpoint. It would not be good policy to

work on the control of abortion only and leave animals in the herd whose product would endanger human health.

5. The work of the Bureau of Animal Industry will be conducted cooperatively with the owner and his local practicing veterinarian.

Work of this nature belongs in the field of the local practicing veterinarian. He is in close contact with the herds in which work is being conducted and is the proper person to cooperate in it. So long as he supports and cooperates with the definite policy, owners should seek his advice. The owner is the chief benefactor and should bear the expense of the local practicing veterinarian.

6. The history is obtained and blood is collected from all animals in the herd, both male and female, by the owner's practicing veterinarian, under the supervision of the Bureau of Animal Industry and tested by said bureau.

The history pertaining to abortions which have occurred in the herd, animals which have been added, etc., is important for determining the time for the next blood-test and helps explain the reason for certain percentages of reactors, and is an important basis for future references to the herd.

After the herd has been negative and all reactors have been removed, it is not necessary at each blood-test to test animals under one year of age, or animals that are not old enough to be bred. They should be blood-tested, however, before being bred and all animals in the herd should be blood-tested once a year, or at the regular annual blood-test.

7. A list of cattle blood-tested shall be supplied the bureau at each time a test is applied, so that each blood sample can be identified by the name, date of birth and registry number of the animal and tattoo or ear-tag number.

Complete and permanent identification is very necessary for checking one blood-test against a previous test. Lack of proper identification each time a blood test is made causes more difficulty in carrying out the plan than anything else encountered. Each time a blood-test is made a complete list of all cattle in the herd, and all that have been removed since the last blood-test, should be supplied. Notations should be made on all animals not included in the test, giving reasons why not blood-tested. If this information is not supplied, it is impossible to check the list of animals against a previous list.

8. In a herd in which there are reactors to the first blood-test, all reacting animals shall be either disposed of in a manner satisfactory to the Bureau of Animal Industry or placed on separate premises. The original premises must be thoroughly cleaned and disinfected.

The plan has been most successfully carried out where reactors have been disposed of or removed immediately following test.

9. In a herd in which reactors are found, as described in section 8, a retest should be applied, in thirty to sixty days subsequent to the date of first blood-test, and additional reactors should immediately be disposed of and removed, as provided in section 8.

In herds where reactors are disclosed on any blood-test, the tests should be repeated every two months until all animals remaining in the herd have been negative for a period of one year, after which time it is advisable to blood-test at least as often as two or three times each year. Bulls as well as females should be blood-tested. If the disease is active at the time the plan is put into operation, tests more freqeuntly will aid in getting ahead of it, even every thirty days. In herds where all animals are negative to the initial blood-rest, and a subsequent sixty-day test, the tests need not be repeated any more frequently than in herds that have been negative for one year.

10. The herd shall be blood-tested at such intervals as deemed advisable by the Bureau, but in no case shall there be a period of more than one year between blood tests. If additional reactors are found they shall be handled as provided in section 8. Individual animals shall be subjected to such blood-tests or retests and at such intervals as the Bureau of Animal Industry deems advisable, for the effective working of the plan.

Occasionally the Bureau deems it advisable to make frequently repeated blood-tests on individual animals which have shown a suspicious or uncharacteristic test or something connected with the history may indicate more frequently repeated tests. Occasionally, due to some defect in the method of carrying out the plan, it is deemed advisable to determine same, and make frequent blood-tests, in order to make headway in controlling the disease.

11. Any animal which aborts in any herd under supervision must be immediately isolated and reported to the Bureau of Animal Industry. The place where the abortion occurred must be immediately cleaned and disinfected; the fetus and membranes must be disposed of properly.

Occasionally abortions occur as a result of causes other than Bang bacillus, but all should be handled as Bang bacillus abortions until proper studies have been made to determine that they are not. The cow should be kept in isolation until these studies have been completed. Her blood should be tested two weeks or later, or at least before she is again bred, after the abortion occurred. A negative test within two weeks after the abortion should not be depended upon. In any event she should be kept in isolation until her discharges following the abortion have ceased. The fetus and membranes should be sent to the Bureau laboratory, when possible, for study; also a sample of her first milk. The fetus and membranes should be so disposed of that no cows or other animals can some in contact with them.

12. Herd bulls must not be used for service in other herds and cows from other herds shall not be brought into them for service.

Herd bulls may be used from one certified "Abortion-Free" herd to another, provided in transportation they are completely protected from exposure to disease as provided in this plan, and provided this practice does not interfere with the tuberculosis-eradication requirements.

13. All milk and other dairy products fed shall be either produced by an approved herd or shall be adequately pasteurized.

14. Cattle from approved herds may be added without a blood-test. If shipped, the car must be cleaned and disinfected and public stock-yards must be avoided.

15. In no case shall pregnant animals be added to a clean herd unless coming from an approved abortion-free herd. Pregnant animals, other than those from approved herds to be added, must be isolated until after having calved and must have passed a blood-test not within two weeks after having calved.

Pregnant animals occasionally do not react their first time until after having aborted or calved apparently normally. Some breeders who were attempting to carry out the plan have failed in this respect and by so doing have prolonged the time of obtaining an abortion-free herd. This is an important recommendation in the plan.

16. All cattle, both male and female, other than those as provided in sections 14 and 15, to be added, must have passed a blood-test approved by the Bureau of Animal Industry, must be isolated for a period of at least sixty days and must have passed the second test applied in accordance with section 6.

17. Cattle removed from the farm for exhibition or any other purpose shall be provided with separate quarters and shall not be exposed to cattle other than those from approved abortion-free herds. If shipped, section 14 must be complied with.

18. The premises must be in a sanitary condition. After the removal of affected animals, the stable must be properly cleaned by owner and disinfected under official supervision.

19. If the owner desires it, a certificate will be issued for herds in which no reactors have been found over a period of one year and which the history indicates are free from Bang bacillus disease, provided the above requirements have been complied with and the following agreement has been properly signed and executed.

CONTRACT AND AGREEMENT

Between....., Owner,
and the Pennsylvania Department of Agriculture,
Bureau of Animal Industry.

The said Department and Bureau, having presented to me a standard plan for the prevention, repression and eradication of Bovine Infectious Abortion (Bang bacillus disease), in the interest of protecting and conserving the health and value of my cattle,

I hereby certify that I have considered carefully the provisions and recommendations of said plan, and

I hereby agree to employ a competent practicing veterinarian, at my expense, to act under the direction of said Department and

Bureau in applying the precautions and measures prescribed in said plan, and

That I will observe and comply with the requirements specified therein for the proper operation of the plan with my cattle and premises.

In the event that I fail to comply with the terms herein set forth or with the requirements of the plan, this contract and agreement shall be cancelled, at the option of said Department and Bureau.

(Date)..... ....., Owner.
(Witness)..... (P. O. Address).....

My entire herd is comprised of the cattle enumerated below:

.....Cattle	.....Purebred	.....Grades	..... Total
Females over 1 year of age			
Females under 1 year of age			
Males over 1 year of age			
Males under 1 year of age			
Total			

Predominating Breed.....
Location of Farm.....
Township.....County.....
I desire to employ Dr.....
whose address is.....

The purpose of the plan is to assist breeders, through their local practicing veterinarian, in getting rid of Bang bacillus disease; to help them in this way to place their herds on a more profitable basis, and to establish herds from which animals free from Bang bacillus disease can be purchased. The certificate is merely evidence that the plan was carried out until the herd qualified.

The following certificate is used:

Certificate No..... Issued.....

Commonwealth of Pennsylvania

DEPARTMENT OF AGRICULTURE
Bureau of Animal Industry
Harrisburg, Penna.

Certificate of Inspection and Test for
BOVINE INFECTIOUS ABORTION

This is to certify that the entire herd of.....
.....P. O.,.....
County, Pennsylvania, consisting of.....
cattle, has undergone inspection; has passed the required number

of blood tests; the owner has complied with the requirements prescribed by the Bureau of Animal Industry for the prevention, repression and eradication of Bovine Infectious Abortion; and the history indicates the absence of infection with Bovine Infectious Abortion. The above-mentioned animals have been found free from all evidence of Bovine Infectious Abortion and are approved by the Bureau.

.....

Director, Bureau of Animal Industry.

This certificate is good for one year from date issued unless revoked.

SUPPLEMENTS—JANUARY, 1927

20. Bull clubs should be handled the same as a single herd consisting of units. Each unit must comply with all requirements of the plan. Bulls which have been in infected units or units that have not qualified for a certificate may enter non-infected units only as added animals, as defined in section 16. It is advised that different bulls be used in infected and non-infected units. No unit will be given a certificate until all members have qualified.

21. Young calves from reacting dams should be kept separate from the negative herd. In no case should calves come in contact with feed or forage for the herd.

22. In a herd holding a certificate, if one reactor to the blood-test is revealed and there is no visible evidence of spread of the disease, the reactor shall be removed and the certificate shall be held in suspension. If at a subsequent blood-test in not less than thirty days, no additional reactor is revealed the certificate shall no longer be suspended.

23. If in any herd holding a certificate more than one animal reacts to the blood-test or, if one (or more) animal aborts and is infected with Bang bacillus, as determined by blood-test or otherwise, the certificate becomes void and the herd must requalify.

24. In Pennsylvania, Bang bacillus disease (bovine infectious abortion) is now a reportable disease. All reactors, whether in herds being operated under the Pennsylvania Plan or not, must be properly reported to the Pennsylvania Bureau of Animal

Industry, Harrisburg, Pennsylvania, and should be handled in accordance with the Bang bacillus disease regulations.

In Successful Operation

The Pennsylvania Plan for prevention, control and eradication of Bang bacillus disease has been successfully carried out during the last six years and its feasibility need no longer be doubted. It is voluntary on the part of the owner but is available to all Pennsylvania cattle-owners who wish to take advantage of it. We believe that the execution of its principles constitutes the only successful method of prevention, control and elimination of this disease which has thus far been produced.

The number of herds operating under the Pennsylvania Plan has been gradually increasing ever since its inception. Up to the present time, approximately twenty-five herds have received "Abortion-Free" certificates and a number of others will soon qualify. Blood-testing on abortion is being done in approximately 500 additional herds.

During the year 1926, approximately 20,000 blood samples were tested for Bang bacillus disease by the Pennsylvania Bureau laboratory. With the exception of the blood examined in connection with the few herds that are under direct Bureau supervision for experimental purposes, the samples were collected and submitted to our laboratory by practicing veterinarians.

The Bureau insists that the local practicing veterinarian participate in this work at the owner's expense, for the reason that the practicing veterinarian is qualified or can qualify more readily than any person who does not have veterinary training. The work belongs in his field and the owner is the chief benefactor and should bear the expense of his services.

In a number of instances local practicing veterinarians and agricultural extension representatives have cooperated to a mutual advantage.

Inquiries regarding the Pennsylvania Plan for preventing and controlling Bang bacillus disease have been received from breed societies, calf clubs and bull associations, which indicates keen interest in this disease on the part of these organizations.

The Jefferson County Fair, at Brookville, Penna., adopted a rule prohibiting the exhibition of cattle unless blood-tested and free from Bang bacillus disease. As far as we know, this is the first fair association to have taken such advanced steps with

reference to the prevention and control of Bang bacillus disease. The buildings were placarded as follows:

ALL CATTLE EXHIBITED
AT THIS FAIR
ARE TESTED AND FREE FROM
CONTAGIOUS ABORTION

ABORTION REACTORS

The question often arises, "What is done with the reactors from Bang bacillus-infected herds?" This question perplexed us some for the reason that Bang bacillus disease in Pennsylvania had not been proclaimed transmissible and was not reportable until September 10, 1926, when it was proclaimed a transmissible disease. The plan did provide however that reactors must be disposed of in a manner satisfactory to the Pennsylvania Bureau of Animal Industry but the plan is voluntary and the owner's own conscience was usually the deciding factor in the disposal of reactors. They were usually disposed of, however, to a butcher for immediate slaughter or in some other manner satisfactory to the Bureau.

Some breeders claim to have valuable animals of merit, yet assert that they cannot afford to separate the diseased from healthy animals. We believe there are no breeders who have valuable assets which they cannot afford to protect. The infected animal can not be looked upon as an asset.

Since the disease was proclaimed transmissible, the Bureau has authority to regulate the movement of these reactors.*

CONCLUDING STATEMENTS

During the last few years, Bang bacillus disease has apparently become more prevalent throughout the United States. This can be attributed to several causes. The use of living-culture vaccine in some sections has undoubtedly been the cause of some spread of the disease. The extensive tuberculosis eradication campaign has necessitated the restocking of many herds, which in most

*A copy of the proclamation was included in the report of the Committee on Bovine Infectious Abortion of the United States Live Stock Sanitary Association, in the Proceedings of the thirtieth (1926) annual meeting, published in the JOURNAL, March, 1927, pp. 874-879.

instances has been done without the use of precautionary measures against other diseases. Many herds are completely wiped out on account of tuberculosis. In restocking such farms the owner and his veterinarian should see to it that the acquired cattle are free from Bang bacillus disease as well as tuberculosis.

The development of successful methods of animal disease prevention, and control and eradication is a duty of organizations constituted by the law for that purpose and in their development, full cooperation of the local practicing veterinarians should be sought. Such successful developments subsequently means more sound and useful veterinary practice.

Preventing and controlling animal and poultry disease is essentially a veterinary problem. In no other profession or vocation can men be found who, by training or experience, can plan, direct or carry out so efficiently as can veterinarians, the work incident to studying, preventing and controlling transmissible animal diseases.

I have the utmost confidence in the ability of the veterinary profession to solve and otherwise handle satisfactorily Bang bacillus disease. It is a work for which veterinarians have been specially trained and equipped.

Veterinarians and breeders can render the future live stock industry no greater service than by taking steps to place the future industry on a healthy basis tending towards higher efficiency. Veterinarians are confronted, almost every day, with opportunities to use their influence in establishing herds on a healthy basis.

Those practicing veterinarians, particularly in dairy and breeding sections, who lack interest in Bang bacillus disease should become keenly interested in the subject in order that they may properly and profitably advise and guide their clients.

Many herd-owners have shown a greater interest in the development of Bang bacillus disease prevention and control work than many veterinarians. In some instances owners have found it difficult to find in their locality practicing veterinarians sufficiently interested in the disease to cooperate in its prevention and eradication. Veterinarians frequently are not as well informed regarding Bang bacillus disease as breeders and dairymen themselves. This causes a tendency on the part of herd-owners to seek other sources for information on the subject. Many veterinarians are prone to seek cures or some quick, easy method for controlling Bang bacillus disease. This state of affairs should

not exist and would, in a large degree, be prevented if veterinarians were to keep posted by the study of available literature, on the most recent developments relative to the prevention, control and eradication of transmissible animal diseases. Moreover, too many veterinarians confine their attention to the sick animal. They overlook the importance of the physical condition of the other animals, health history of the entire herd and sanitation. To furnish the best service to the client and thus make the greatest success of practice, the veterinarian must consider the herd as a unit rather than the individual animal.

The local practicing veterinarian is the proper person for breeders and dairymen to look to for advice regarding not only Bang bacillus disease but all matters relating to animal health and sanitation. In turn the local practicing veterinarian should be fully equipped and sufficiently interested to furnish this service. Many practitioners are doing this and to a mutual advantage. Far too many, however, are not, and it is the latter group of practicing veterinarians who complain most when breeders and dairymen seek advice regarding health and sanitation from other sources. Disease prevention is essential to a successful and prosperous live stock industry.

A distinguished leader in the medical profession has said: "Prevention of disease is the chief concern of the medical science and that the great goal of the medical world is positive prevention." A prominent teacher at one of our leading veterinary schools has recently said: "Animals are receiving the same scientific care today as human beings and that preventive veterinary medicine is becoming important."

Practicing veterinarians who have not awakened to the necessity of supplying a service of disease prevention to their clients are not only hurting themselves but are jeopardizing the profession. Interest is lacking among far too many members of our profession as to the need of the average dairyman and breeder. Breeders and dairymen realize the necessity of healthy live stock and it is apparent that they will secure the veterinary service they need elsewhere if our profession will not satisfactorily supply it.

Local practitioners will be consulted and employed in connection with the prevention and eradication of Bang bacillus disease in proportion to the value of the service they render.

To prevent and eradicate Bang bacillus disease, successfully, the owner and his veterinarian must be absolutely honest with each other and the state. They must also be persevering. If

either is dishonest or compromising in his methods, they will surely fail in their efforts. The longer I am engaged in transmissible disease prevention and eradication work the more impressed I am with the essentiality for every person participating to be absolutely honest, have a definite plan and stick to it.

The toll from preventable live stock diseases is appalling. The loss of a calf or death of a mature animal from a preventable disease is a challenge to the veterinary profession. It therefore behooves every veterinarian, regardless of the phase of veterinary work in which he is engaged, to take a keen and intelligent interest in carrying out and promoting a transmissible animal prevention and control service. Any local practicing veterinarian can, if he only will, supply such a service, supplemented and assisted as needed by the central control agency, as is being done in Pennsylvania.

We recognize that much remains to be learned regarding Bang bacillus disease. Additional knowledge on the subject is needed and further research and scientific study conducted along intelligent lines is imperative. Authorities are now convinced that the bacillus of Bang is the most important factor in bovine abortions. Some claim that feed deficiencies are an important factor. While we favor conducting research investigations sufficient to determine the relationship of faulty nutrition to bovine abortion, we believe it our duty to continue to deal with the subject as a transmissible disease proposition.

In Pennsylvania we are endeavoring to apply in the field what information we possess to the best advantage. We are depending on our own laboratory, as well as research workers elsewhere, to continue the scientific study of Bang bacillus disease and keep us informed regarding new knowledge acquired.

Bang bacillus disease is being prevented and eradicated in our state by the Pennsylvania Plan. While the plan may not be perfect, it is undoubtedly the best one thus far produced. We commend it to you with the thought that you, like ourselves, will use it to the best advantage and in so doing will learn more about the disease and wherein the plan needs changing and strengthening. In other words, practice and make perfect the best we have.

We are greatly interested in the success of your efforts and wish you well in studying, preventing and controlling Bang bacillus disease in Illinois.

CARNIVOROUS FEEDS AND FEEDING*

By RUDOLPH H. SCHNEIDER, *Boston, Mass.*
Angell Memorial Animal Hospital

The business of being a worthwhile veterinarian is a serious business, and requires that we think and do worthwhile things. Correlated with and inseparable from our professional duties are our educational obligations to owners of animals. Our mission is primarily that of treating sick animals—to cure them of their ills, in the shortest possible time. To do so we must exercise every possible means at our disposal. Thus, we find it is not merely a matter of diagnosis and prescription-writing, but includes such accessory measures as the regulation of the diet, hygienic control, and the like.

I have chosen as my subject a discussion of the feeds and feeding of carnivorous animals, because I believe it is a key whereby we can make ourselves worthwhile and instructive in a subject that is scantily understood, hence leading to much harm and confusion. What we need is a common standard, the facts of which I hope to bring forth in this paper, so that eventually dog-owners may learn, through you all, to feed the proper foodstuffs as religiously as they now avoid them.

The feeding of animals, both in health and disease, constitutes no little portion of our armamentarium in avoiding and combating sickness. In fact, so vital is it, I believe we could get along fairly well with it alone, both as a preventive measure and a curative agent. We all realize that a large portion of the indispositions of dogs are traceable to faulty feeding and a good many cures can be wrought by the institution of proper dietetics. As an example I will cite a case brought to my attention by the owner of a pack of hounds.

One after another of his dogs developed "fright disease" or "running fits," until all of the pack became afflicted with these periodic seizures. What was the cause and how was it to be corrected? I directed him to discontinue the feeding of dog breads and put them on a diet of raw beef, milk, green vegetables and stale whole-wheat bread. Soon after this change the trouble ceased. But this is not all, for I was soon to learn that the amelioration was only temporary. Questioning revealed that

*Presented at the monthly meeting of the Massachusetts Veterinary Association, March 23, 1927.

he was feeding his dogs essentially the same as I had directed but that he had again found a new plan of economy, and was feeding ground refuse bread, procured from hotels, instead of the stale and dried whole-wheat bread. Examination of samples revealed that the bread had fermented and soured. The previously suggested diet was again instituted, and soon order was once more restored, now permanently, for there has been no further trouble in a year's time. This is only one instance of many in which the diet was the causative as well as the curative agent.

In this particular malady, fright fits, I am thoroughly convinced that the trouble can be traced to the stomach, in most instances. Not solely because of the feeding of dog breads and other prepared foods, though they play an important role in this respect, but from other indigestible foodstuffs, such as improperly prepared carrots, boiled liver, and the like. Such acute cases are promptly alleviated by the administration of an emetic. It is to be remembered that similar seizures are often premonitory to the onset of other maladies, notably distemper.

Eczema Caused by Faulty Diet

Unquestionably, a large portion of our eczema cases are likewise brought about by a faulty diet. There are differences of opinion relative to the type of food conducive to this trouble. I am convinced that the carbohydrates are in a large measure prone to produce the disturbance in dogs, due to their heating proclivities, while in cats it is most often due to continued feeding of canned salmon or cooked liver. However, I am also reasonably sure that in either case the lack of fresh, uncooked food plays an important role.

We are all acquainted with the appalling prevalence of malnutrition we find in the majority of young dogs—emaciated, rickety, spiritless, and what not. Why? Principally because they have been improperly fed. The staff of life—meat and milk—is denied them. Dog breads have supplanted the old stand-bys. Sure enough they like them. Yes! They would probably like shoe leather if that was all they got to eat. If a dog develops fits, the owner will promptly advise you that the patient stole a meat ball and she is sure that it is at fault. If so, it is probably because the pup became hysterical from the delight afforded by such a rare bit. Such indigestible foods are no more fitted to them than the same would be to an infant

baby, and who would think of feeding a young child baked biscuits, even though properly balanced?

This is only a trifle, compared with the vast amount of medication they receive in a vain effort to overcome the indispositions caused by their feeding. Is there any wonder that they fall prey to diseases? I often wonder that there are not more that succumb to distemper. There is constant talk of specific medication for this malady, but what could we expect if we had it? Certainly not much, in the type of dogs that are reared now-a-days. In this deplorable state of affairs we can and should make it our duty to do much missionary work. To me it seems well spent energy to take the time to explain to owners, at every available opportunity, the importance of properly feeding these little waifs. You yourselves will eventually benefit on future calls, when, in all likelihood, you will find a dog that has not only built up a body but a resistance behind it that will help you in your efforts to restore him to order, from whatever may be the indisposition.

Again, there is a form of apostematous dermatitis, a scurvy-like affliction, and a form of paresis in dogs, both of which can be traced to faulty feeding. These will usually be found in dogs continually fed on cooked food and those lacking in certain necessary vitamins.

Natural Foods Desirable

The small animals we have to deal with most, belong to the classification—carnivora. As the name implies—flesh-devouring—the essential part of the diet consists of the proteins, and derived from the animal kingdom. Although a good many of these animals can adapt themselves to non-carnivorous foods, as readily as they have to their unnatural mode of living under domestication, the fact still remains that the majority will do much better on their natural foods, as they would also were they allowed to live in the manner that most becomes them.

All foodstuffs are essentially the same, in that they contain all the elements necessary for growth and function of the animal body, whether they are derived from the animal or vegetable kingdom, but they are not similarly digestible in all animals. In carnivorous animals the major part of digestion is gastric, while in the herbivora, the intestinal functions are paramount, the stomach functions being mostly preparatory in character. In omnivora we have a combining form of digestion. By reason

of the dog's intended diet, there is little need of salivary digestion and he bolts his food. Foods then that require this form of digestion are therefore more or less harmful to the dog as he will likewise bolt them as he does his meat. If fed they should be given in a form that will require mastication. In herbivora, on the other hand, mastication is of vital importance, hence the lengthy function. Correspondingly, the other internal organs are relatively larger and smaller, depending upon their requirements.

In the dog, as in other animals, there is need of the carbohydrates and fats, but in a lesser degree, hence the lesser necessity for a long intestinal tract. These foods are the fattening and heating elements for the animal economy. There is little radiation of heat and penetration of cold through the skin and haircoat of the dog, and it may be that that is why they require less of these elements in their rations. Under natural circumstances carbohydrates are derived from the glycogenic content of the muscles and liver consumed, while the fat, of course, is obtained from the adipose tissue eaten.

Dogs Must Have Meat

You will see from what I have said that a totally animal diet is complete in itself for all the requirements of the carnivorous organism, and you will realize how intricately complete it is in every essential before I am finished. Our aim must be to duplicate this natural diet in adapting a mode of feeding. Circumstances are materially different, yet the ration must be essentially the same. In the cat we have less to worry about as long as we allow him some freedom and do not overfeed him, for he will supply himself with some unimprovable meals, derived from the carcasses of the small rodents and birds he preys upon. The dog, however, requires especial attention. Fundamentally, as stated, he must have meat. At least half of his diet should consist of this element. An abundance of meat during the growing period without question produces a body more resistant to disease and conducive to a greater come-back after infection. Lean flesh has practically no fattening powers and we may look to it as a good article of diet in obese patients. It is satisfying, yet letting the animal derive its carbohydrate and fat from his own economy until a proper balance and condition are restored.

Beef is our most natural and best selection for meat supply, although for the sake of variety and to meet certain necessities

lamb, horse flesh and fresh fish may be substituted. Not only are the fleshy structures of the carcass to be thought of, but also the glandular and internal organs, as the heart, pancreas, liver and kidneys. However, it would be far better to adhere strictly to the feeding of the muscular parts than the glandular elements; though both are essential, the latter being particularly rich in vitamins. The best form in which to feed meats, *especially the glandular forms, is raw.*

Fish, if fed to the dog as a meat substitute, had better be boiled, so that the bones may be picked out before being fed. If cooked meats are fed, they had better be roasted, boiled or broiled but *not fried.* For the cat, fresh uncooked of parboiled fish, raw beef, and *uncooked liver* are suitable. Canned, smoked, salted, spiced and dried meat preparations, as well as pork, are taboo and should never be fed the dog or cat, as they are decidedly detrimental to their well-being. Fish is readily digestible yet not so nourishing as other meats. As with other foodstuffs, idiosyncrasies must be taken into consideration, as certain types of fleshy foods may be ill borne by certain individuals, while taken with impunity by others. This is more particularly true of fatty foods. As a rule, however, meat should be fed with the fat that comes with it. Always remember that there are more dogs suffering for want of meat than there are those that have suffered, or ever will, from its consumption.

Large Raw Bones Are Important Part of Diet

Large raw bones should always be allowed the dog. The gnawing of them improves the condition of the teeth, as well as supplying the animal with an additional supply of the necessary mineral salts, such as calcium, phosphorus and iron. These are especially necessary for the metabolism of the puppy. As a rule the dog can grind up and digest small bones with impunity, but as they sometimes lodge in the alimentary tract, they had better not be recommended. Muscular meats are notably deficient in vitamin content, but liver, heart and kidney are abundantly supplied with these vital nutritional and metabolic elements. Liver tops the list, with the others following in the order named. Raw eggs are a useful but expensive substitute for meat. They should not be forgotten, however, in nourishing the sick.

Let us now consider the carbohydrates as an element in the ration. They require little of our time, as they do not essentially

interest us. They are the starches. They are derived principally from the vegetable kingdom and although they help to balance the ration, they are not generally suitable for the dog and cat, from the standpoint of digestibility.

Among the most acceptable vegetables for the dog are tomatoes, spinach, lettuce, carrots and onions. They contain an abundance of all the known vitamins, so essential in every diet. This fact can also be taken advantage of in the treatment of certain deficiency diseases. With the exception of carrots, these vegetables may be fed raw or cooked, but carrots must be mashed and boiled, else they are likely to cause serious digestive disturbances. As for the cereal grains, whole wheat is undoubtedly our best selection, cornmeal, barley, polished rice and whole rice being quite unsuitable. They are however, all rich in growth-producing vitamins. Whole wheat is best fed in the form of a stale whole-wheat bread, preferably oven-dried as well, to avoid any chance of heating through fermentation.

Milk One of Our Greatest Foods

Milk is one of our greatest foods, its main drawback being its excessive bulk. However, its virtues cannot be overlooked, especially in the young. It is a perfectly balanced foodstuff, containing all the necessary principals for proper development and is easily digested and assimilated by both young and old. It is rich in calcium and phosphorus and the three principal vitamins. For puppies it is all-sufficient for their needs, but soon has to be supplemented with raw meat, to keep pace with their development. Because of its high standard, it is a wonderful adjunct to other foods, both for its relish and high food value. There are some dogs that cannot tolerate whole milk, principally because of the aversion to its fat content. Such cases might better be given butter-milk, which is equally as wholesome as the whole milk but for the absence of fat. It also has the advantage of being more readily digested and, like sour milk, has been known to expel intestinal parasites. Milk is of course indispensable as a food for the sick, either alone or with eggs.

Although in the animal's natural state milk is fed only the young, through the dam, there is no good reason why we should not feed it to the dog as a regular part of his daily ration, as we do the cat. It makes a dainty dish for the mature dog who receives but the one substantial meal a day. It is best fed in the morning, with the hearty meal at night. No dog could ask

for a more suitable ration than one consisting exclusively of milk and meats. Evaporated and condensed milks are not so valuable as the fresh whole milks, though relished by some individuals. Of the two, the evaporated is to be be preferred, as it contains vitamins A and B and a little C. The condensed is entirely devoid of these elements.

Vitamins Must Be Considered

The vitamin content of the various foodstuffs requires much consideration in selecting a diet for the dog. This is especially true during the growing age but must never be lost sight of. Just what they are we do not know, except through their manifestations. They are practically the most vital of all food factors. There are three important vitamins, namely, A, B and C, and several others. Vitamin D really is intimately associated with vitamin A.

Vitamin A is fat-soluble and is known to insure against rickets and eye troubles, the latter property being considered a possible due to vitamin D. The fat-soluble vitamin is included in milk, butter, eggs, cod-liver oil, beef fat, glandular organs and leafy vegetables.

Vitamin B is water-soluble and is known as the anti-neuritic vitamin and has to do with growth. Its absence leads to dwarfishness, anemia, stilted gait and even paralysis. Yeast, nuts and grain germs are richest in this type of vitamin. These foods are not so suitable for the dog, as they are not readily digested. However, there is an abundance in liver. In addition there is a goodly supply found in heart meat, kidneys, lettuce, carrots, tomatoes, oranges, whole and evaporated milk.

Vitamin C is also water-soluble and is known as the anti-scorbutic vitamin. It protects against skin troubles and unhealthy teeth. It is included in milk, a few vegetables and the citrous fruits. There is a plentiful supply, however, in suitable dog foods. Liver, heart and kidney all contain vitamin C, liver again topping the list. Muscle meats are devoid of this element, as are the fats and oils like cod-liver oil, butter and also eggs. Whole milk and skim milk contain a small quantity. However, there are some suitable vegetables that are rich in this vitamin. Tomatoes, spinach and lettuce rank high, with carrots and onions rating lower in the scale. Oranges are also rich in this vitamin and although it is not thought of as a dog food it is useful in cases presenting a deficiency of this element. Every one would

do well to acquaint himself with these vitamins and where they are found in most abundance, for they are the where-with-all in the successful treatment of deficiency disturbances so common in our young dogs.

Feeding should occur at regularly stated times, as it becomes a habit with the dog, and at the time of anticipation digestion is at its best. Puppies should be fed little at a time and often, while the mature dog in health should do well with but one substantial feeding per day. No hard-and-fast rule can be made as to the quantity of food a dog should receive, as this varies as much with different individuals as it does with the various breeds and the work required of them. The amount can be determined only by observation. A safe guide is to feed the least quantity necessary to be consistent with proper condition and functioning.

ANOTHER SWINDLER AT WORK

One or our members located in Cincinnati reports an experience with a new variety of swindler who looks upon professional men as his particular prey. This party goes under the name of Dr. Grim and he claims to be manager of the Medical-Dental Adjustment Bureau. Several months ago, this party opened up a collecting agency in Cincinnati and called upon physicians, dentists and veterinarians in that city, for the purpose of having them place past-due accounts in his hands.

The veterinarian who reports this case placed several hundred dollars worth of old accounts in Dr. Grim's hands and on two occasions the latter returned to the veterinarian with checks which he had collected and which were made payable to the veterinarian The checks were brought to the latter for endorsement and amounted to over $60.00.

About the first of April, the veterinarian called at Dr. Grim's office, only to find that the latter had been called away very suddenly "on sickness" and up until May 12 had not returned. Upon investigation it was found that a number of local physicians were included among the victims, the total number of whom is not known. The party traveling under the name of Gr. Grim is about fifty years of age, five feet five inches in height, has gray hair and is usually well dressed. It is more than likely that this party will turn up in other cities and veterinarians are warned to be on the lookout for him.

AN APPARENT HEREDITARY EPITHELIAL DEFECT FACTOR, THE POSSIBLE ETIOLOGY OF BURSATTEE IN HORSES

By C. E. HOWELL *and* G. H. HART

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A predisposition to sensitiveness and irritation affecting the epithelium of the skin of some horses and resulting in a more or less extensive pathological alteration during certain seasons of the year has been observed in various parts of the world. Different names have been applied to the malady, depending on local conditions which were thought to have a bearing on its etiology. Thus, in India, the term bursattee was applied to it from the Indian word, burus, meaning rain, because the disease appeared during the hot, sultry, rainy season. In Florida it was described under the name of leeches, on account of its appearance in animals grazing in lakes and swamps infested with leeches. Horses entered these ponds at times to such a depth that their backs were submerged. In Texas the term sandburn has been used. In that state, near Falfurrias, the disease was studied by one of the writers (G. H. H.)[16] in 1908. The trouble usually appeared on hot sunny days after a rain. Certain low-lying areas of land where rain water collected were thought by the stockmen to be particularly dangerous and in some cases such areas were fenced off from the range to keep horses out.

Equine mycosis has been used in referring to it on account of fungi, particularly the fusarium fungus, being isolated from the affected areas. Summer sore is the common name used throughout this country, as it develops in animals during the summer season and they usually undergo complete recovery, except for the remaining scar tissue during the cold seasons of the year. Hutyra and Marek,[17] under the names of malignant hyphomycosis, leeches and bursattee, report only a single case having been observed in Europe and reported by Drouin and Rénon, in Alfort. However, under *Filaria irritans* a more extended discussion is given and work by Railliet, Descazeaux and others mentioned.[18] It is also stated that unsightly scurfing of the skin in so-called summer sores was frequently observed during the World War in the months of June, July and August, when flies

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were present in greatest numbers. The disease exists in France, because importers of Percherons have mentioned to one of the writers (C. E. H.) seeing it there and imported stallions from France have been found affected. In 1850, the disease was reported in France by Bouley,[4] under the name of "Dermite Granuleuses," and appeared only during the hottest summers, disappearing with the approach of cold weather. He, therefore, concluded that extreme hot weather was its primary cause. In Germany apparently little investigation of the disease had been made. During the World War it became common in the German army horses, probably due to conditions under which the animals were kept, and the bedding, feed, particularly molasses, and other doubtful factors were considered as possible causes.

History of Previous Studies

The affection was studied in the early part of the last century before the days of bacteriology and when knowledge regarding many pathological conditions was on an empirical basis. It is not surprising, therefore, that various students of the trouble have tried to associate it with carcinoma, sarcoma, keloid, Delhi boil, farcy, furunculosis, mycosis, and nematode infestation.

It was probably first described in 1829, by Kerr,[19] of the British Army, stationed in the East Indies, who thought it was a poverty disease. The natives believed it was frequently communicated through the medium of flies. A very interesting review of the work during the last century was made by Fish,[11] in a report of the disease in Florida under the name of leeches. Tombs,[33] of the Bengal Artillery, in 1831, found some persons in India who believed the origin of bursattee could be traced to Arab blood. He stated:

> Horses of a mungrel breed are highly susceptible to this disease; horses whose sires are Arabians and dams country bred mares. The Honourable Company's stud-bred horses are most of all subject to it. English and thoroughbred country horses are least of all liable to it. I should not omit to mention here that we have several English thoroughbred stallions imported annually; country-bred horses are covered by them, the offspring of which are more prone to this disease than any other race of horses in India; this I can prove by facts.

His conclusion was that the predisposing cause may have been foreign blood, at least foreign to India, and the exciting cause vicissitudes of temperature. This is a very significant statement, in the light of the studies here reported.

In 1872, Hart,[15] of Calcutta, studied the disease and considered it to be non-infectious and peculiar to the tropics. He divided

the cases into three forms, depending on their gravity as papillated, medium, and phagadenic bursattee ulcer. Involvement of the lymph-glands he did not find to occur except in bad cases. Histopathological study failed to reveal anything like cancer, but he felt the condition was allied to this affection. He tried several inoculation experiments to transmit the disease but with negative results.

F. F. Collins,[7] of Bombay, although he believed it a parasitic trouble, stated: "In horses that once show a bursottee diathesis an ordinary wound is alone sufficient to establish the disease."

"Max,"[23] in 1876, considered bursattee to be nothing more or less than an ordinary wound aggravated by climatic conditions. He supported the fly theory of infection. An editorial in the *Veterinary Journal* of the issue in which "Max's" article appeared pointed out that after ulceration had disintegrated the surface, mycelia or other low forms of organisms may be present but this is not the essential cause but rather an accidental introduction from without. He also mentions in quotation from the Veterinary Aide Memoire that where there is a constitutional predisposition to the disease any accidental rub or wound may turn, during the hot weather and rains, to bursattee. A statement is also made that one of the most experienced practical judges of horses in India felt he could prove that bursattee is hereditary.

Meyrick,[24] in 1878, in India, reported a case of a high-caste Arab stud, fifteen years old, in which pieces of kunkur as large as a pea or bean were deposited under the whole pleural surface of both lungs. He also found two or three pieces of kunkur on the lungs in another old Arab horse which had never shown any signs of bursattee. It is probable that these findings were in no way related to bursattee. The term kunkur is applied in India to a prevalent form of limestone used in road-making. In old-standing cases of bursattee, concretions are sometimes found in the areas of stony hardness composed mainly of lime salts and to them the name kunkur was also applied.

Smith,[31] in 1879, carried on efforts to transmit the disease to animals and to man. The latter experiment subjects included ten native grooms, one native farrier and himself. The results were all negative, although in one horse and one human subject there was some induration about the inoculated areas. One of the lines along which he suggested research should be carried out was: "Statistical records as to hereditary transmission of the

disease, prevalence in sex and breed, influence of age, temperament, and general management."

Burke,[6] of the Dick Veterinary College, was strongly of the opinion that the trouble was cancer, despite the previous work of Hart, which failed to demonstrate anything like cancer structure histologically.

Smith,[32] in 1884, described a mould fungus in fresh specimens but it could not be demonstrated after hardening in chromic acid. He further stated that he had lately produced typical bursattee ulcers by inoculation with portions of the tumor and intended to endeavor to produce the same condition with the cultivated moulds.

In a communication to the U. S. Bureau of Animal Industry, J. C. Neal,[25] of Archer, Florida, in 1887, described leeching. He stated: "All horses do not leech, only those of good blood. The Cuban and Texan ponies, as a rule, are exempt. It is not contagious. The mule is rarely affected, cows still more so."

A. W. Bitting,[3] of the Florida Experiment Station, in 1894, made an investigation of the disease and from pathological study thought it was a malignant tumor of the round-cell sarcoma variety. He, however, considered the influence of heredity. While but few records were obtained, the inference was drawn that it did have some effect, although environment for successive generations may have made the offspring more susceptible.

In southern Europe and northern Africa the trouble was confirmed as identical with bursattee of India by Fleming.[12]

The first finding of parasites in the areas was made by Ercolani,[10] in 1859. He applied the name of *Trichina uncinata* to the nematode found and regarded it as the causative factor. In 1868, Rivolta[29] found a worm, which he termed *Dermafilaria irritans*, to be quite regularly present in the sores. His findings in the southern Europe cases were confirmed in 1884 by Laulanie.[20] The complete life cycle of the parasite was not known. Law[21] states:

> The presence of the worm in the center of the caseous debris of the lesion implies that it is the essential factor of causation. It is not, however, easy to explain the fact that but one horse in a stable suffers year after year from the local lesions when once started, without any tendency to implicate other horses standing alongside or working with it in the team. It would seem as if the disease should be cut short by the death or elimination of the larval worms, and that the other horses in the same stable, subjected to precisely the same environment, should also contract the affection.

At another point, when discussing the local propagation of a cryptogam as the causative agent, the following statement is made:

> Equally difficult is it to explain why in the more temperate regions, like New York, the disease will persist for years in one horse in a stable and respect the second horse of the same team, though dressed daily with the same brush and rubber and pestered by the same flies that suck the liquids from the kunkurs. Manifestly as in many cases of favus and ringworm the one animal acquires a constitutional susceptibility to which the other is a stranger.

In 1914, Lewis,[22] while chief veterinary officer for the northern territory of Australia, carried on extensive experiments with a skin affection termed equine granuloma or swamp cancer. He considered this trouble in the main to agree with leeches in Florida, as described by Fish. Microscopic study of lesions failed to reveal any parasites but eosinophilia was present in the sections. Efforts to transmit the affection in various ways, especially by applying leeches from the ponds of the area to normal horses, after they had fed on the summer-sore areas of affected horses, all resulted negatively. Considerable evidence was presented to show that flies are not involved in its transmission.

In 1924, Nielsen,[26] of Denmark, reported studies made of the malady in Denmark. It was first described in that country by J. H. Selmer, in 1902, at a meeting of the Fyns Veterinary Association, at Odense. The latter originally described the ulcers under the name of "raadsaar," but soon applied the generally accepted term of summer sores to them.

Ransom,[28] in 1913, described the life cycle of one of the Habronema species (*Habronema muscae*). He showed that the adult stage of this parasite was located in the stomach of the horse. The embryos passed out with the feces and the intermediate host was the ordinary house fly (*Musca domestica*). The embryos entered the fly larvae and underwent their various stages of development with the developing larvae. They are usually found in the head or in the proboscis of the fly. These partially developed parasites are deposited on the lips of the horse or may gain access to the stomach by the horse consuming flies with feed. The adult female nematode in the stomach of the horse is 13 to 22 mm. long and 250 to 400 microns thick, the male being somewhat smaller. The work of Descazeaux,[9] presented at the French Central Veterinary Society by Railliet, in 1915, describes summer sores in Brazil, and gives illustrations of the parasite found in the lesions. Railliet and Henry,[27] in

considering the work of Ransom and Descazeaux, came to the conclusion that the parasite was the etiological factor and gained access to the summer sores by means of flies.

It can readily be understood, in case of infestation of horses with these parasites and flies as the intermediate carriers, that larval forms of the worm could readily be deposited in the ulcers of summer sores and become encysted. There is no desire on our part to show that Habronema larvae may not cause certain abnormalities in horses, such as conjunctivitis and a granulomatous condition of the prepuce, as reported by Bull,[5] in Australia. They may also be a factor in enhancing the seriousness of summer-sore areas to which they gain access, without in any way being a primary factor in their origin. Railliet, however, was of the opinion in 1915 that the larvae could penetrate the skin of the horse when, together with manure, they come in contact with it.

Van Saceghem,[34] in 1917, at the Zambi station in Belgian Congo, observed that the horses were badly infested with *H. muscae* and summer sores were also common. He found that infested flies coming in contact with previous abrasions in the skin caused the infestation with the larvae. In his experiments, however, the Habronema larvae were not able to penetrate the skin of a horse as suggested by Railliet. When he placed the larvae from flies on the skin of a horse, they soon died, but when placed on an abrasion or ulcer they quickly buried themselves in the tissues. He carried out one other interesting experiment. On an experiment horse he produced two abrasions. One was covered with a bandage, while the other was left unprotected. The horse was then placed in a stable where previous investigations had shown 20 per cent of the flies carried Habronema larvae. The protected wound healed in a normal manner, while the unprotected wound developed into a typical summer sore.

Data have recently been published by Freeborn, Hart and Howell,[13] showing that summer sores exist among the Percheron horses on the University Farm in the complete absence of the Habronema infestation.

Recent Genetic Studies

While a number of early investigations of this trouble suggest the possibility of heredity being involved in its etiology, the science of genetics had not developed at that time. Comparatively recent work, particularly in human beings, has shown that defects of various kinds are transmitted through families.

The inheritance of defects in domestic animals has been given little attention because of the prejudice against breeding defective individuals. When defects appear in animals these defective individuals are very seldom bred. They are culled out of the herd.

The inherited defects may be due to either dominant or recessive factors. Among the examples of dominant factors Babcock and Clausen[1] mention: keratosis, thickening of the epidermis; epidermolysis, excessive formation of blisters, hypotrichosis, a hairless and toothless condition; and piebaldness, spotting of the dermal coat. Recessive factors are alkaptonuria, multiple sclerosis, degeneration of upper part of the spinal cord and hereditary ataxia. One example of a sex-linked recessive factor of an inherited nature is hemophilia.

Hadley and Warwick,[14] in 1926, reviewed the inherited defects in live stock. The former made a study of a peculiar congenital epithelial defect in calves in which the following description of the lesion is given:

> Defective formation of the skin below the knees and hocks; absence or incomplete development of one or more claws; deformed ears due to rolling of the margins and growing together of the contacted surfaces, which shows that these lesions antedated birth by some weeks; defects in the integument of the muzzle; defective areas in the mucous membranes of the nostrils, tongue, hard palate and cheeks.

These animals were never able to live longer than a few weeks. The forty-three cases found were in thirteen different herds all located in Wisconsin and traced to the same foundation stock, imported from Holland in 1871.

Warwick[35] has experimental evidence to show that inguinal hernia in swine is a heritable character. A hereditary lethal deformity in new-born lambs has recently been reported by Roberts,[30] in Scotland. It is thus seen that as studies on heredity are extended, evidence accumulates that domestic animals as well as man are subject to a great variety of defects which are transmitted genetically.

University Farm Horse Breeding Operations

The University Farm was established in 1906. It has always been the practice to keep the Division of Animal Husbandry horses, which have largely been purebreds, separate from the Farm Division horses and mules which were grades of unknown breeding. There are at present in the Farm Division barn twelve horses and sixteen mules. The Animal Husbandry stud at the present time comprises a total of thirty purebred horses, of

which one is Standardbred, five are Thoroughbreds, four are Shires and twenty are Percherons. The Standardbred mare has been on the farm since 1910. The Thoroughbreds were first introduced in 1921, when the Remount Division of the U. S. Army placed the stallion, Gunrock, here for service. This was followed by the acquisition, in 1924, of two mares and the other two are get from these animals. Four other foals from these two mares have been sold from the University Farm.

While four grade Shires were in the stud from 1910, the first purebred was obtained in 1915, when a prize-winning mare was obtained at the Panama Pacific Exposition. A Shire stallion was loaned to the Farm in 1916 and three pregnant imported mares were brought here to foal and to be rebred to this stallion. A filly and a stallion foal from the imported mares were donated to the Farm. The present Shires consist of the mare purchased at the Panama Exposition and offspring of the foals donated in 1916.

History of the Percheron Horses at the University Farm

This is the only breed in which summer sores have developed. Although one purebred Percheron mare was on the Farm from about 1913, the foundation stock of Percherons was purchased in May, 1916, from the Paicines Ranch Company, Paicines, California, which shortly thereafter disposed of their entire stud. It consisted of five two-year-old fillies, two yearling fillies, and the stallion, Fernand 72763 (5262). The seven fillies were sired by Fernand. Those foaled in 1914 were Fermante of Paicines 112093, Paule of Paicines 111453, Ferette of Paicines 111454, Ferlotte of Paicines 111452, and Pauvane of Paicines 111451. The two foaled in 1915 were Fernand's Luceile 122864 and Fernand's Lottie 122865. A brief history of each one of the animals of the original purchase follows.

The mare Pauvane of Paicines was a full sister of Fernand's Luceile, i. e., she was sired by Fernand and out of Luceile. She developed the most extensive summer sores of any animal at the University Farm. The condition was more or less general on her legs and body with a large ulcer on the left front pastern. She was valueless as a work animal and after exhaustive efforts to remedy the trouble failed, she was finally destroyed, June 21, 1921. She produced two foals at the University Farm. One, a filly, Silverleaf 154813, foaled in 1919, developed summer sores.

Silverleaf was sired by Aiglonel 121004, a stallion purchased in 1917. On account of the summer-sore affection, we did not wish to keep Silverleaf as a purebred brood mare and she was sold without her registration papers. The purchaser was fully cognizant of her condition and said he would take her to the coastal region where the climate was cool during the summer months and he was sure the mare would not show the skin eruptions. Two years later he reported that the mare became so badly affected that she was destroyed. The mare Pauvane of Paicines was bred a second time to Aiglonel and produced a stallion foal in 1921. He was castrated and when three years of age was sold. He did not show the condition while at the University Farm and his present condition is not known.

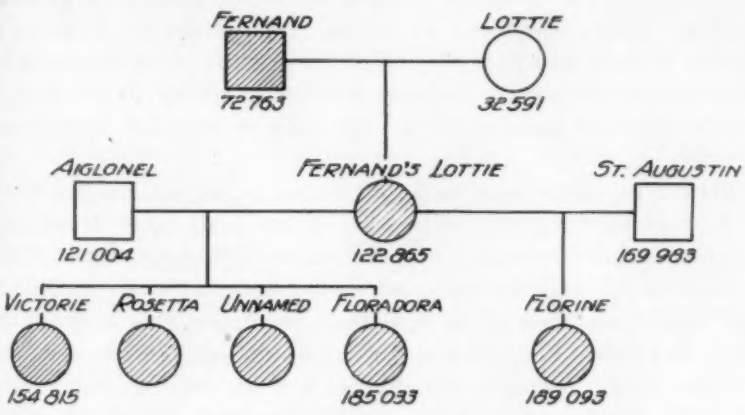

FIGURE 1. A diagram showing the family history of Fernand's Lottie. Squares designate stallions and circles indicate mares. The shaded symbols indicate the animals personally examined by the authors, and which showed clinical evidence of summer sores. The name of the animal is given and the number refers to the registration number of the animal in the Percheron Society of America.

Ferlotte of Paicines was sired by Fernand and out of Lottie 32951, thereby being a full sister to Fernand's Lottie, the mare that has summer sores and whose colts all have the defect. Ferlotte of Paicines never showed the defect. She was bred twice to Aiglonel and produced two stallion foals, one in 1919 and one in 1920. Both of these colts were castrated and neither of them ever exhibited any evidence of the defect. Ferlotte of Paicines died, April 29, 1921, with metritis following abortion.

Paule of Paicines was out of a mare called Paicines Pink 56094. Four colts were produced by Paule of Paicines. Three of them were sired by Aiglonel. One was sired by a purebred Shire stallion (accidental breeding). None of these animals has shown

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While four grade Shires were in the stud from 1910, the first purebred was obtained in 1915, when a prize-winning mare was obtained at the Panama Pacific Exposition. A Shire stallion was loaned to the Farm in 1916 and three pregnant imported mares were brought here to foal and to be rebred to this stallion. A filly and a stallion foal from the imported mares were donated to the Farm. The present Shires consist of the mare purchased at the Panama Exposition and offspring of the foals donated in 1916.

History of the Percheron Horses at the University Farm

This is the only breed in which summer sores have developed. Although one purebred Percheron mare was on the Farm from about 1913, the foundation stock of Percherons was purchased in May, 1916, from the Paicines Ranch Company, Paicines, California, which shortly thereafter disposed of their entire stud. It consisted of five two-year-old fillies, two yearling fillies, and the stallion, Fernand 72763 (5262). The seven fillies were sired by Fernand. Those foaled in 1914 were Fermante of Paicines 112093, Paule of Paicines 111453, Ferette of Paicines 111454, Ferlotte of Paicines 111452, and Pauvane of Paicines 111451. The two foaled in 1915 were Fernand's Luceile 122864 and Fernand's Lottie 122865. A brief history of each one of the animals of the original purchase follows.

The mare Pauvane of Paicines was a full sister of Fernand's Luceile, i. e., she was sired by Fernand and out of Luceile. She developed the most extensive summer sores of any animal at the University Farm. The condition was more or less general on her legs and body with a large ulcer on the left front pastern. She was valueless as a work animal and after exhaustive efforts to remedy the trouble failed, she was finally destroyed, June 21, 1921. She produced two foals at the University Farm. One, a filly, Silverleaf 154813, foaled in 1919, developed summer sores.

Silverleaf was sired by Aiglonel 121004, a stallion purchased in 1917. On account of the summer-sore affection, we did not wish to keep Silverleaf as a purebred brood mare and she was sold without her registration papers. The purchaser was fully cognizant of her condition and said he would take her to the coastal region where the climate was cool during the summer months and he was sure the mare would not show the skin eruptions. Two years later he reported that the mare became so badly affected that she was destroyed. The mare Pauvane of Paicines was bred a second time to Aiglonel and produced a stallion foal in 1921. He was castrated and when three years of age was sold. He did not show the condition while at the University Farm and his present condition is not known.

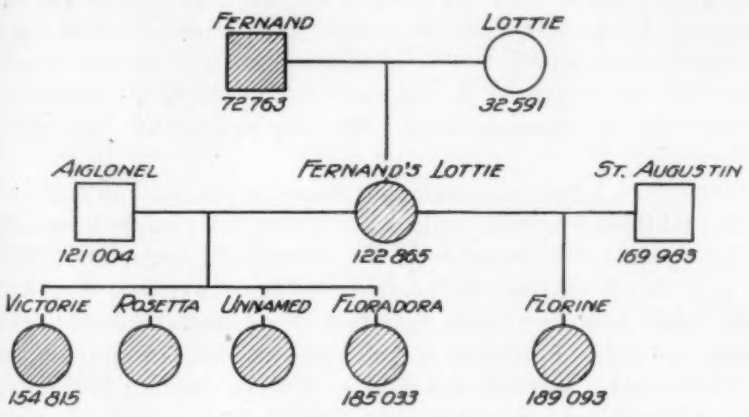

FIGURE 1. A diagram showing the family history of Fernand's Lottie. Squares designate stallions and circles indicate mares. The shaded symbols indicate the animals personally examined by the authors, and which showed clinical evidence of summer sores. The name of the animal is given and the number refers to the registration number of the animal in the Percheron Society of America.

Ferlotte of Paicines was sired by Fernand and out of Lottie 32951, thereby being a full sister to Fernand's Lottie, the mare that has summer sores and whose colts all have the defect. Ferlotte of Paicines never showed the defect. She was bred twice to Aiglonel and produced two stallion foals, one in 1919 and one in 1920. Both of these colts were castrated and neither of them ever exhibited any evidence of the defect. Ferlotte of Paicines died, April 29, 1921, with metritis following abortion.

Paule of Paicines was out of a mare called Paicines Pink 56094. Four colts were produced by Paule of Paicines. Three of them were sired by Aiglonel. One was sired by a purebred Shire stallion (accidental breeding). None of these animals has shown

evidence of being affected with summer sores. This mare died, March 5, 1924, with internal hemorrhages following parturition.

The filly, Fermante of Paicines, died, June 2, 1916, from verminous colic (strongylosis). Ferette of Paicines is at present being used as a chore horse for the stock barns on the University Farm. She was bred only once and produced a stallion foal in 1919. This colt was castrated and sold as a work horse in 1922. It has been impossible to get any trace of this animal and his present condition is not known. He showed no epithelial defects up to three years of age, when he was sold.

The two mares, Fernand's Luceile and Fernand's Lottie, with their offspring, form the basis for most of the investigation on bursattee.

Figure 1 shows that the stallion Fernand, an animal having summer sores, was bred to Lottie and produced Fernand's Lottie, a mare which developed summer sores quite extensively over her entire body. Records are not available to show that Lottie ever had summer sores. She may or may not have been affected.

Fernand's Lottie was bred four times to the stallion Aiglonel and produced four filly foals. All of the foals later developed summer sores. She was bred a fifth time to St. Augustin 169983, a stallion of entirely different breeding, purchased in 1922. She again produced a filly foal that developed the defect. Of these five fillies, Floradora and Florine are being retained in the College herd. Victorie was sold as a work animal, November 16, 1922. Rosetta was used in an experiment in which the effect of mouldy silage on horses was tried. She survived the experiment but later died (November 30, 1922) from impaction. The fifth unnamed filly was foaled, February 12, 1922. She was such a poor type, either as a work horse or breeding animal, that she was sold, August 14, 1923.

Figure 2 shows the breeding operation carried on with the mare, Fernand's Luceile. This mare was bred to Aiglonel and she also produced four filly foals, as did Fernand's Lottie. One of them, Vanity, developed summer sores. The other three were free from any clinical evidence of the defect. Fernand's Luceile was bred to St. Augustin and gave birth to a fifth filly, Princess, which has not developed the condition. Bred to the same stallion again, she produced twin stallion foals, March 10, 1926. The twins are not old enough to ascertain whether or not they will develop the summer-sore condition. The offspring of Fer-

nand's Luceile have been kept at the University Farm, except Lucretia. She was sold, January 10, 1925, to a Percheron breeder of the Sacramento Valley, living about twenty miles from Davis. The mare has been observed many times by one of the authors (C. E. H.) since she was sold and does not show any evidence of summer sores.

Discussion

The foregoing data show that despite the various breeds of purebred and also grade horses and mules which have been maintained on the University Farm since its establishment in 1906, only the foundation stock of Percherons purchased in 1916 and their offspring have shown evidence of summer sores. We have also shown that Habronema infestation does not exist

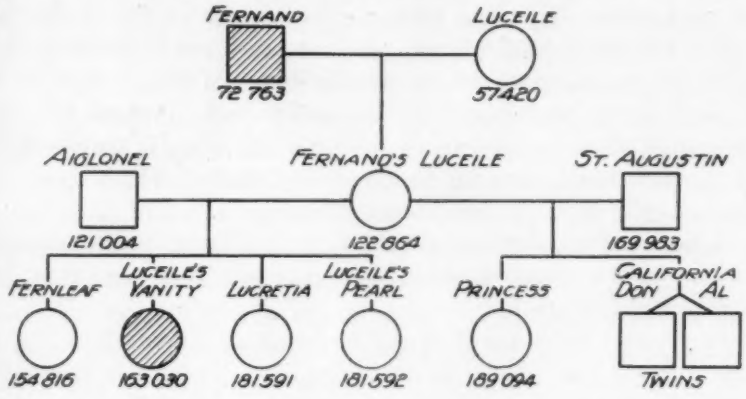

Figure 2. A diagram similar to figure 1, showing the family history of Fernand's Luceile.

in the horses on this farm at the present time. No effort has been made to separate affected from normal animals. They have been kept in the same barn and adjoining corrals. Affected and unaffected horses have worked in the same teams. The horses have been fed alike at all times. The condition has developed on pasture as well as in stall-fed and corral-fed animals.

The only explanation we have to offer, therefore, for the selective manifestation of this trouble on the University Farm is that its primary cause is a family characteristic produced by a hereditary factor in the particular family of Percherons.

The fact that all of the foals of Fernand's Lottie have shown the defect, even when bred to stallions of entirely different lineage, is evidence that this mare is homozygous for this factor. Fernand's Luceile bred to the same stallion has produced only one

affected mare, which indicates that she is heterozygous for the factor.

Figure 1 could be explained on the hypothesis that the factor for summer sores is dominant. All of the offspring from a homozygous mare crossed with two apparently normal stallions produced heterozygous foals, all showing the defect. However, in figure 2 Fernand's Luceile, although she did not show the defect, was able, when bred to Aiglonel, to produce one affected foal in the four offspring. If it were dominant we would expect Fernand's Luceile to show summer sores if she could transmit them. This chart, therefore, can be more easily explained on the theory that the factor is recessive, both the stallion, Aiglonel, and the mare, Fernand's Luceile, carrying a recessive factor which in Vanity became homozygous and the defect appeared. Such an explanation is in line with our knowledge of the factor for red in Aberdeen Angus cattle. In this breed red is recessive and must be homozygous to be manifested. Many of the black animals carry the heterozygous recessive factor for red but red color in this breed appears only when an individual is the offspring of parents both carrying the recessive factor, which becomes homozygous in one-fourth of the offspring.

A hereditary condition in barley, requiring an environmental factor for its manifestation, has recently been reported by Collins,[8] of this station.

In a barley field near Berkeley he found an albino plant. This was dug up and placed in the greenhouse, where it gradually became green and produced seed. Plants raised from the seed in the greenhouse—mean temperature, 65.0° F.—always produced normal green plants. When, however, they were grown outside the greenhouse in December and January—mean temperature, 48.5°F.— and thus subjected to a considerably lower temperature, they produced albino plants. The factor for albinism in the plant was thus hereditarily transmitted, but became manifested only when the environmental factor of cool weather was present.

It is quite possible that in summer sores there is more than one factor involved in its transmission in addition to the environmental factor of vicissitudes of temperature. In this regard mention might be made of the strains of white sweet peas studied by Bateson and Punnett.[2] These strains of white peas, when bred true, produced all white flowers. When the white strains were crossed with each other it was the usual result to get all

white flowers. In one instance, however, there resulted no white flowers but only purple-colored ones, similar to the color of the wild Sicilian ancestors of the plant. This is explained by the authors that the purple color appears only when two independent dominant factors are present together and that it results from some sort of interaction between them. Unless some such hypothesis is the explanation for the appearance of the trouble, it is hard to understand why summer sores are not more prevalent among horses generally.

Summary

Evidence is presented to show that the prime etiological factor in summer sores is a genetic one. Observations have been made by workers during the past century to substantiate this hypothesis The data are presented in order that, where opportunity offers, information may be accumulated on this question. We do not have sufficient data to give the scientific genetic explanation of the exact nature of the transmission of the defect. Opportunity exists in the stud for further study of the problem, but on account of the time consumed in the reproduction cycle in the horse and the fact that there is usually only a single offspring at the termination of each gestation period, the accumulation of data is rendered exceedingly slow.

Despite the world-wide distribution of the trouble, it has not become of great economic importance in horse husbandry, because of the natural widespread tendency of breeders to eliminate defective animals from the studs.

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MISUSE OF CATTLE TAGS A SERIOUS OFFENSE

A case of much interest to cattle-owners was recently heard in the Franklin County, Pennsylvania, Court of Quarter Sessions.

Beattie Coldsmith, a farmer and cattle-dealer, plead guilty to charges of obtaining money by false pretense. The charge grew out of the misuse of two official ear-tags of the kind used by the Bureau of Animal Industry, Pennsylvania Department of Agriculture, to mark cattle that have passed an official tuberculin test. Coldsmith held a cattle sale on February 3, at which he represented that all cattle had been tuberculin tested and officially tagged. The Bureau of Animal Industry learned that Coldsmith had obtained surreptitiously two identification tags and placed them in the ears of two untested cows and sold them to an unsuspecting purchaser who believed the cows were tested.

The Bureau then brought the charge of defrauding the buyers, to which Coldsmith plead guilty. When passing sentence on the defendant, Judge Davison pointed out that while it may seem a slight offense to misuse a tag, the offense had grave possibilities and that the extreme penalty for the offense may be $500 fine and three years' imprisonment.

At the request of the Bureau representative who made the complaint against Coldsmith, the Court suspended sentence for two years after ordering the defendant to pay $50 and the cost of prosecution before October 1, 1927.

INVESTIGATIONS OF STEPHANURUS DENTATUS (KIDNEY WORM) OF HOGS

Preliminary Report

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Stephanurus dentatus is one of the most destructive parasites among swine in the South. It ranks very close to the Ascaris in economic loss to the swine industry in this section, due to the fact that the parasite attacks animals of breeding age and interferes materially with the future development of the herd.

According to Hall,[1] the male is 2.0-3.7 cm. long by 1.25 mm. wide; the female is 2.5-4.5 cm. long by 2.0 mm. wide. It is a thick, mottled worm, having a semi-transparent integument, intestines convoluted, being much longer than the body of the worm. The tail of the female is curved ventrally, is obtuse and possesses a conical tip. The caudal extremity of the male presents a bursa which is inconspicuous and almost rectangular. The cephalic extremity of both male and female is blunt.

In the host the parasites are found most abundantly in the sublumbar region, chiefly in the perinephric tissue, and along the course of the ureters. They are often present in other portions of the body, including the lungs, pleural cavities, diaphragm, liver, kidneys, spinal canal, peritoneal cavity, sublumbar muscles and the sublumbar mesenteric attachment. The presence of the kidney worm gives rise to numerous parasitic cysts and abscess formations, varying in size from 0.5 to 4.0 cm. in diameter. Within these cysts are usually found a male and a female kidney worm, although the presence of four or five parasites may be seen in the same cyst. The parasites may likewise occur within the recently formed abscesses. However, in the old formations they are not present. The pus within the abscess is of a greenish-yellow color and upon a microscopic examination shows the presence of various pus-producing organisms and eggs of the parasite. The parasitic cysts and abscesses are located principally within the perinephric adipose tissue, in the sublumbar mesenteric attachment, and along the walls of the ureters. When the parasite occurs within the lungs and pleural cavities, such

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occurrence is usually without the characteristic lesions mentioned, but in many cases there is present an extensive sero-fibrinous pleuritis. In its occurrence within the spinal canal, the presence of a slight cystic formation may be seen, and a local edematous

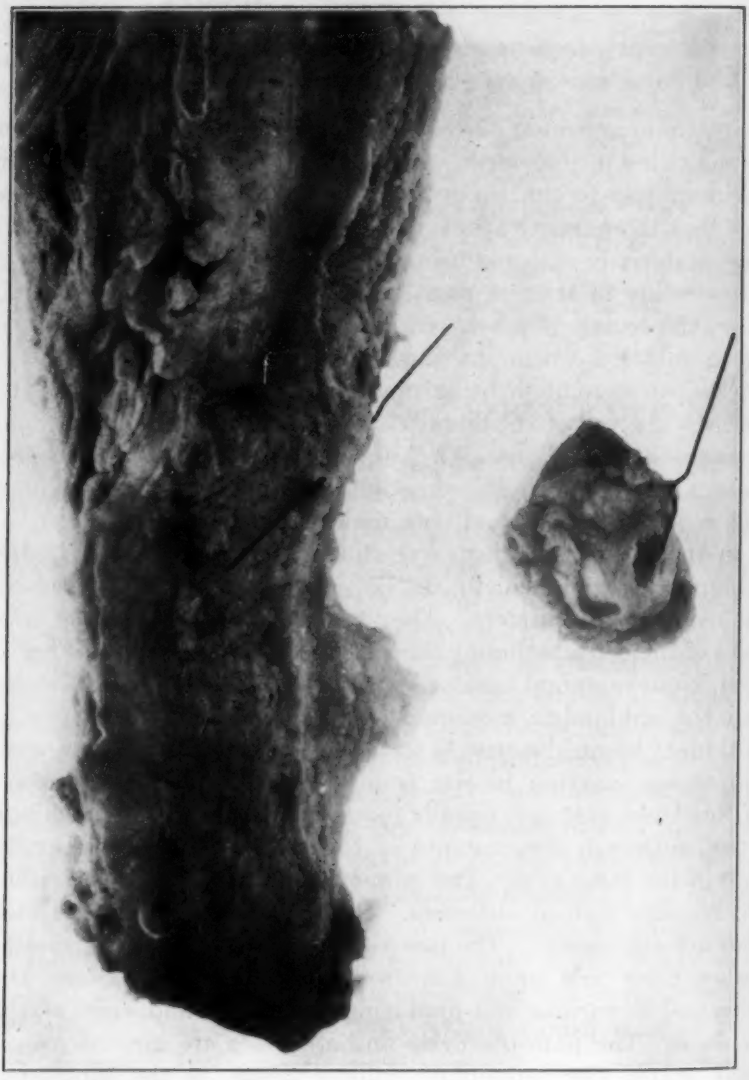

FIG. 1. On left, ureter dissected longitudinally, showing fistulous tract entering organ. On right, cross section of ureter, showing parasitic cyst and fistulous tract.

infiltration of the meninges occurs. We have observed the parasite occurring on the dorsal and ventral surfaces of the spinal cord.

In nearly every case of infestation, lesions are found along the course of the ureters. These lesions consist of the parasitic cysts, containing the parasites. These cysts are very numerous in severe infestation, and they are connected with the lumen of the

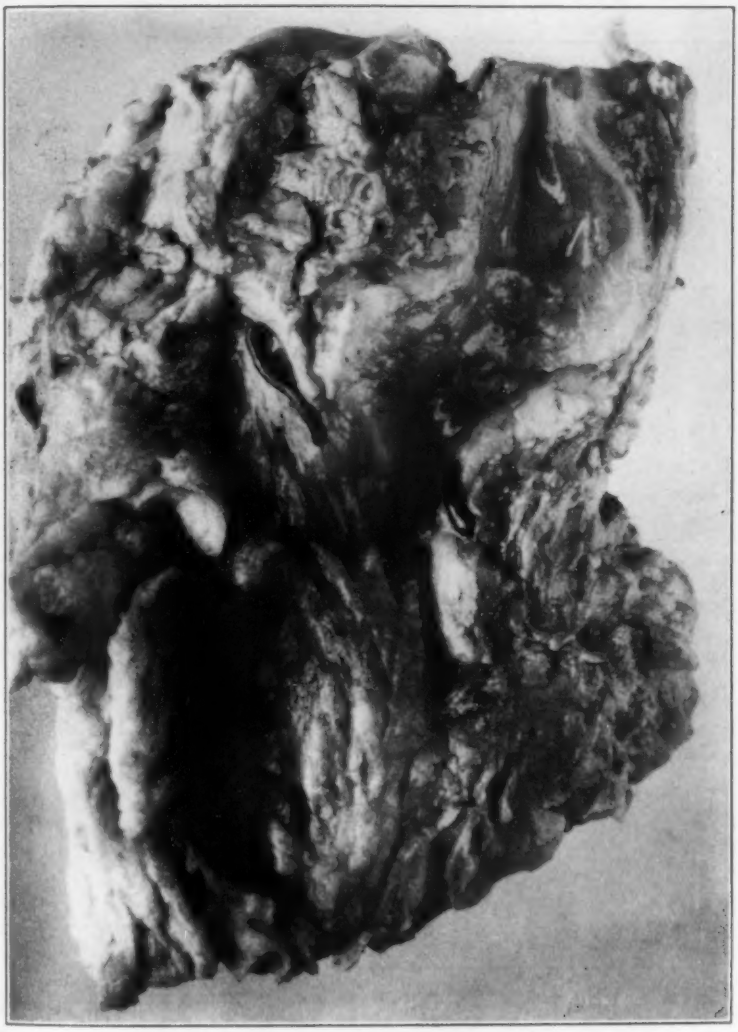

FIG. 2. Ureter dissected longitudinally, showing two fistulous tracts containing parasites, and also showing thickening of wall of ureter, with the tortuous course.

ureter by small fistulous tracts. The parasites may be found in the small fistulous tracts as well as within the cysts. Undoubtedly the fistulous tracts leading from the cysts in which the mature

parasites are found serve as channels for passage of the eggs into the ureter or kidney, and these eggs pass out with the urine. The urine in these cases always contains numerous eggs. When these lesions are found along the course of the ureter, the wall of this organ is greatly thickened and its course becomes tortuous.

FIG. 3. Kidney and portion of ureter, showing small fistulous tracts and two parasites near pelvis of kidney.

In some cases these lesions are so extensive as to produce a partial or complete obstruction of the lumen of the ureter. In such cases, hydronephrosis is produced as a consequence of the impediment in the flow of urine along the course of the ureter. Chronic cystitis is often present, and in such cases the urine

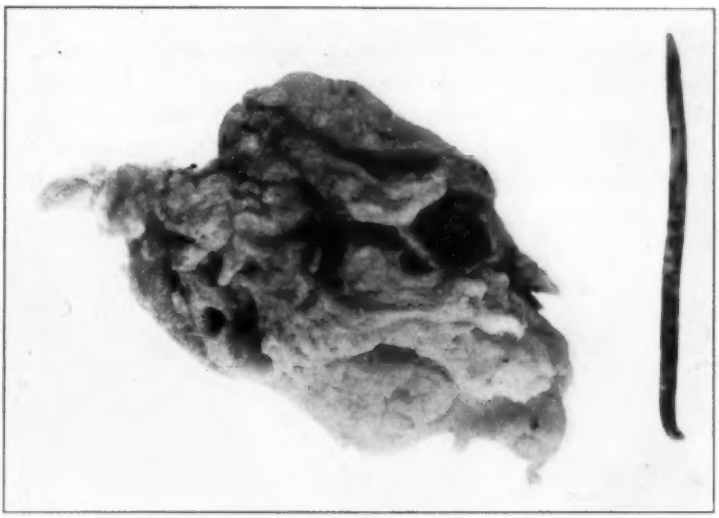

FIG. 4. On left, parasitic cyst, containing parasite. On right, adult female, *Stephanurus dentatus*.

contains a quantity of pus, casts, and eggs of the parasite. The liver is indurated in severe cases and multiple abscess formations are present. Parasites are often found in the abscesses of the liver.

In the advanced stages of infestation abscesses occur in most

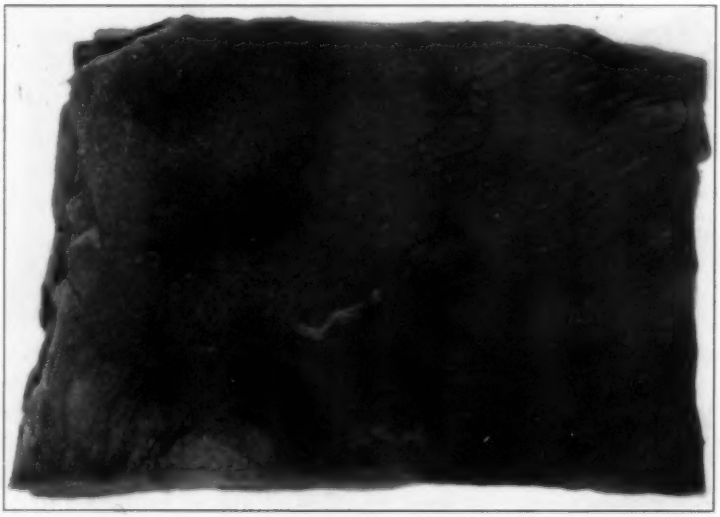

FIG. 5. Lung, showing presence of parasite. Surface of lung shows evidence of sero-fibrinous pleuritis. (Note: There were six parasites found on the surface of the lung from which this photo was taken.)

every part of the abdominal cavity, involving many of the organs in this cavity. In these cases, an extensive adhesive peritonitis, accompanied by ascites, is usually present. There is no perceptible systemic disturbance noted in animals harboring but few of the parasites, but infestation can be detected upon making a microscopic examination of the urine, since the eggs of the parasite may be found in the urine of infested animals even though the animal shows no symptoms of the disease. That moderately infested animals show no ill effects is further demonstrated by the fact that numerous hogs so infested are encountered at the slaughter-houses, yet these animals are in good physical condition and show no symptoms of the infestation.

FIG. 6. *Stephanurus dentatus* occurring within spinal canal. A, parasite; B, spinal cord.

After the degree of infestation has progressed to such an extent as to produce systemic disturbances, there is a loss of appetite accompanied by a loss of flesh and general unthriftiness. Many cases at the onset of the symptoms show a slight incoördination of movement in the posterior limbs, which is followed later by a well-marked paraplegia. When this stage is reached the animal drags itself from place to place by use of its fore limbs. These cases may linger in this condition for weeks before death ensues, and at times they may show an apparent improvement of this paralytic condition. Animals which show this slight improvement cannot withstand much exertion, for exercise causes a complete loss of use of the posterior extremities. It would seem reasonable to believe that this paralytic condition is due to one of two conditions or a combination of them, i. e., the presence of the parasite within the spinal canal with its associated pathological lesions, or the absorption of toxic products arising from the numerous lesions and possibly from the parasites themselves.

The conditions which are most favorable for the infestation of the kidney worm include insanitary hog-lots which have filthy wallows, insanitary places for feeding and watering, and where a large number of hogs are kept continuously on a small area.

Apparently the only way to control the losses occurring incident to infestation with kidney worms is through sanitation and pasture rotation. Medical treatment is of no avail, due to the inaccessible location of the parasites within the host.

At present experiments are being conducted to determine further the exact life history of the kidney worm and its mode of entrance and migration within the body of the host.

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BIG MEETING IN ATLANTIC CITY

The semi-annual meeting of the New Jersey State Veterinary Medical Association will be held in Atlantic City, at the Hotel Chelsea, June 20-21-22, at which time the Association will entertain the Eastern States Tuberculosis Eradication Conference, comprising the federal Bureau of Animal Industry, the New Jersey State Board of Agriculture and the sanitary officials and others interested in tuberculosis eradication from the following states: Maine, New Hampshire, Vermont, Massachusetts, Rhode Island, Connecticut, New York, New Jersey, Pennsylvania, Delaware, Maryland, Virginia and West Virginia.

SOME OBSERVATIONS ON "HOG FLU" AND ITS SEASONAL PREVALENCE IN IOWA

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U. S. Bureau of Animal Industry

The disease of swine, which, for want of a better name, has been termed "hog flu," first made its appearance in Iowa late in the fall of 1918. Perhaps, it would be more correct to say the disease first began to attract attention at that time and was recognized by veterinarians as presenting a different clinical picture from the swine epizootics which had prevailed before that time. Dr. J. S. Koen, who was in charge of the U. S. Bureau of Animal Industry hog cholera control work in Iowa at the time, is generally given the credit for naming the disease and was one of the first to direct attention to it.

The appearance of this disease in Iowa was coincidental with the severe outbreak of human "flu" which swept over the country in the fall of 1918. This coincidence in the appearance of the two diseases and some similarity in the clinical symptoms served to engraft the term "flu" on the new swine epizootic.

Not a few farmers and some veterinarians thought that the two diseases were associated and that the swine might have contracted the disease in the first instance from human beings. Even at the present time, since the disease has become well established in Iowa, that is, since it has become widely prevalent and regularly recurrent each year, it is not uncommon to meet farmers and veterinarians, who believe they have themselves contracted the disease by coming in contact with affected swine. The writer was always inclined to veiw this latter belief with skepticism until a year or two ago, when he had an experience which inclines him to believe that, after all, there may be some foundation for it. This experience may be worth citing, not, of course, as proof, but as merely pointing to the possible communicability of the disease to human beings.

In the latter part of November, 1924, the writer was engaged in the investigation of hog flu and had gone to a small town situated in southeastern Iowa, where the disease was prevailing very extensively in the surrounding country at the time. The writer took with him from the Bureau Laboratory, at Ames,

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apparatus and culture media with a view to securing bacteriological cultures and pathological material. Through the courtesy of the post-office authorities, a temporary laboratory was established in the Federal building, a thoroughly modern, well-heated and well-ventilated structure. Arriving on Sunday, in excellent physical condition at the time, the writer spent the three following days in visiting many farms, where the hogs were suffering from flu in the acute stages of the disease. In going into the hog houses to observe the sick animals, clouds of dust were frequently encountered, when the animals were roused and driven out into the yards. When roused, the sick animals immediately begin to cough violently and the floating particles of dust undoubtedly become contaminated with the fine spray thrown off from the throats and respiratory tracts of the sick animals.

Possibility of Cross-Infection

With fifty to a hundred hogs coughing violently in a dusty barn, the breathing of the contaminated dusty atmosphere by a human being would certainly afford an excellent opportunity for infection, provided, of course, that cross-infection can occur. The writer had intended to return to Ames on Thursday (Thanksgiving Day), but the evening before he suffered from an unaccountable loss of appetite, accompanied by nausea, and during the night had slight fever. It was at first thought that something eaten at a restaurant had caused these symptoms. On the following morning, however, there was aching in the back and joints and the writer remained in bed, returning home the day after Thanksgiving Day. There were no symptoms of acute coryza, but a very troublesome bronchial cough developed, which hung on for a month or more. The cough was paroxysmal in character, resembling whooping-cough, but the writer had suffered from a severe attack of that disease in boyhood and was undoubtedly immune to it. The cough was not troublesome at night, nor when resting quietly, but the paroxysms seemed to be brought on by exertion and, in this respect, was quite similar in nature to the cough which is one of the characteristic symptoms of hog flu. This indisposition was so entirely different from the usual run of colds and was so similar in its sudden onset and in some of its symptoms to swine flu, that the writer has been rather inclined to think it must have been a respiratory infection acquired as a result of breathing the dusty atmosphere of the hog barns occupied by swine suffering from acute hog flu. As far as

he knew, the writer had not come in contact with any cases of human flu, or influenza, and this disease was not prevailing at the time. Of course, this may have been a purely coincident infection, picked up in some other way, nevertheless, as pointed out above, the circumstances seemed to indicate a possible connection with the swine flu, which was being investigated at the time.

The onset of hog flu, as already stated, is sudden, an entire herd coming down, as a rule, within a day or two. Sometimes, the entire herd is simultaneously affected, that is, on the same day, and at other times half the herd is affected on one day and the other half by the following day. The first symptom noted is loss of appetite, the animals failing to come up for their feed. They are disinclined to move and lie around the straw stacks or in their houses. When temperatures are taken, the animals are found to have fever. A thumpy or jerky respiration soon develops which is best observed when the animals are lying down and at first may be so slight as to escape notice unless the animals are carefully watched; later, it becomes more pronounced and may be noted when the animals are standing. The disease evidently has a very short incubation period and develops rapidly. By the second or third day, the entire herd, as a rule, will be lying in their nests and often present a very sick appearance.

Cough a Prominent Symptom

Sometimes, one may walk among the sick animals and even step over them without rousing them, and any one viewing for the first time a herd suffering from hog flu at the height of the infection would probably think that most of the affected animals would succumb. When the sick animals are roused from their nests, they almost invariably cough. The cough is paroxysmal in character, the back being often arched, and the spells of coughing are sometimes of sufficient violence to induce vomiting; in this respect the disease resembles whooping-cough in the human. When the paroxysms of coughing have passed, the animals stand in a listless attitude with their heads down, their tails limp, and soon lie down as though tired. The sick animals usually rest on their bellies, and sometimes assume a partly sitting position with the body propped on the fore legs, as if to afford room for greater lung expansion. There is usually a conjunctivitis, characterized by a watery or gummy secretion from the eyes, and a nasal discharge may also be present. The disease is characterized by a

fever, which, as a rule, reaches the high point by the third or fourth day. The temperature does not usually run quite so high as in acute hog cholera, the average maximum temperature being somewhere around 105 degrees.

The majority of the pigs in a given herd are affected and, as already stated, they usually come down quite suddenly and within a short time of each other. The sick animals, as a rule, go completely off feed and soon become noticeably gaunt; this gaunting-up or loss in weight is one of the characteristic features of the disease. The acute stage of the disease is usually passed within a few days to a week, and there is often a rapid change for the better, which may be almost as marked as the sudden onset. In uncomplicated cases, where the herd receives good care and attention, the mortality is low, ranging from one to three or four per cent, and sometimes there is no loss at all. In cases, however, where the housing conditions are not good and the animals are not properly bedded and are subjected to draughts, in other words, when the sick animals do not receive good care and attention, pneumonia is apt to develop and there may be a considerable loss. The disease appears to be primarily a bronchitis, from which the majority of the affected animals recover with proper care, the fatal cases being due to a terminal pneumonia, probably secondary in character.

Onset Is Sudden

In view of the sudden onset of the disease, which is a respiratory affection, and the fact that a majority of the animals in a herd come down almost simultaneously, it would seem that the etiological factor must be some organism which the animals harbor in the respiratory tract under normal conditions and this organism takes on pathogenic properties when the resistance of the animals is lowered. The true etiological factor, however, has not yet been determined.

While the mortality is low and the animals may be sick but a short time, there is nearly always a marked shrinkage or loss in weight. This loss in weight constitutes the main economic loss and is a very serious one to the farmer, inasmuch as the disease seems to prevail chiefly among shoats weighing around 150 pounds, at about the time that, or only a comparatively short time before, the animals are to be shipped to market. The loss in weight following an attack is usually quite marked and may be out of all proportion to the short time the animals have been

sick. Furthermore, it takes quite a while to replace this loss in weight.

In the fatal cases, the most pronounced lesions, and in fact about the only lesions, are seen in the lungs, which show a patchy or lobular type of pneumonia. The trachea and the larger bronchi are usually filled with a thick, tenacious and sometimes bloody mucus. The lymphatic glands lying along the trachea are, as a rule, greatly enlarged and congested. One attack of the disease confers little, if any, immunity, for it seems to be quite well established that the same herd may suffer two or even three attacks in one season, and the season during which the disease prevails is a short one, as will be explained later.

Seasonal Prevalence

It was early observed, in Iowa, that swine flu is a disease which prevails during a certain season of the year, that is, during the fall and early winter. It was also noted that the appearance of the disease seems to be connected with the approach of cold weather in the fall. It was further noted that the appearance of the disease varies from year to year. In the case of an early fall, especially when characterized by cold rains or early snows, the disease seems to make its appearance earlier, whereas, in the case of a late fall, characterized by mild, dry, and open weather, the appearance of the disease is delayed. In Iowa, the disease seems to develop first in the northern counties, with the somewhat earlier advent of cold weather, and appears somewhat later in the southern counties, following the seasonal change from summer to fall or early winter. There is usually a marked subsidence of the disease by the end of December, but scattered cases may occur during January; by the end of February, the disease has pretty well run out.

On the supposition that the development of the disease is brought about through a lowering in animal resistance, the most plausible explanation of the appearance of the disease with the onset of cold weather in the fall would seem to lie in the fact that during the summer months swine become accustomed to sleeping out in the open, frequently around straw stacks, and seem to be very loath to go into their houses even after cold weather has set in. The writer has often noted herds affected with flu that were still sleeping out around straw stacks in the late fall, during very cold and wet weather, rather than seek shelter in their houses. In such cases, the loss is apt to be high from terminal pneumonia.

The rapid subsidence of the disease between the first and the middle of January may be due in part to the fact that the hogs have become accustomed to sleeping indoors and have also become acclimated, as it were, to the cold weather, but is probably due in large measure to the fact that a large part of the hog population has gone to market by the middle of January.

Even though the hogs may seek shelter in their houses when cold weather sets in, the houses are often so poorly constructed the animals do not have adequate protection from the cold. In many cases there is no system whatever of ventilation in the hog-houses or else the ventilation is very faulty and the animals are subjected to cold draughts. In the matter of bedding the animals, farmers are apt to be grossly negligent. The bedding is frequently insufficient and in cases where the animals sleep in houses or sheds which are also used by cattle, a rather thin layer of straw scattered over a foot or two of damp manure is often considered sufficient. Even where hog-houses of the more modern type have been built, the ventilation of such structures is often very inadequate and faulty. It is a well-known physiological fact that the heat-regulating centers in the hog are not stable and the animal is very subject to extremes of both heat and cold. This fact, however, is not fully understood by the farmer and probably accounts for the poor treatment that swine are apt to receive at his hands in the matter of housing and bedding. There are also, unfortunately, many farmers who actually believe that hogs will thrive best amid filthy surroundings, which accounts for the wide prevalence of parasitic and intestinal diseases in these animals, resulting in a lowering of the animal resistance and an increased susceptibility to other diseases.

Hygienic Surroundings Important

If swine are kept in well-constructed, well-ventilated houses, which are free fron draughts, and are given plenty of good, clean bedding, which is changed from time to time and kept dry and free from dust, and the animals are driven into these houses at night upon the first appearance of cold weather and for a time thereafter until they become accustomed to sleeping indoors, it is more than probable that the animals will go through the flu season without contracting the disease.

There is no specific treatment for hog flu. However, if the affected animals receive good care and attention at the start and are provided with good sleeping quarters, free from draughts,

and good bedding, and are put on a light diet, there will probably be little if any loss. Drugs or drug compounds, which are supposed to act as respiratory disinfectants, are usually prescribed by the practicing veterinarian and may or may not be of some benefit.

Since its first appearance in the fall of 1918, hog flu has recurred in Iowa with unfailing regularity and always in the fall of the year. For several years after it first appeared the disease attracted considerable attention from farmers and also from veterinarians. The farmer, being confronted with a new swine disease characterized by sudden onset and often apparently grave symptoms, was naturally alarmed and sought the services of his veterinarian. Later on, when he found there was no specific treatment for the disease and the sick animals would recover with little or no loss provided they were given good care, the farmer then ceased to call in his veterinarian. At the present time the disease has become so well recognized the farmer is able to make his own diagnosis and seldom consults his veterinarian. The latter fact makes it very difficult, in fact, practically impossible, to obtain any definite and reliable data in regard to the prevalence of the disease at the present time, or in recent years, as compared with earlier years. In the earlier years, when the veterinarians were busy treating or attempting to treat the disease, more or less definite data could be obtained through them and the disease was then certainly widespread. It is probable that the disease is still quite prevalent in Iowa during the late fall and early winter months. The extent to which it prevails seems to vary somewhat from year to year.

Infection Probably Not Spread By Fairs

In explanation of the seasonal recurrence of hog flu, the idea has been advanced that the disease is started by the return of show hogs to farms after they have developed the flu at fairs. In order to determine whether there was any foundation for this supposition, the writer made a careful investigation of fairs held in different sections of Iowa during the fall of 1925. As a result of this investigation and from information gathered from practicing veterinarians and others, it appears that show hogs which contract flu at fairs may communicate the disease to hogs on the farms to which they return, but it does not appear probable that the widespread epizootics of swine flu which have been observed in the past originate in this manner. In Iowa, the

State Fair and practically all of the county fairs are held during August and September and the extensive flu outbreaks on farms do not come, as a rule, until a later date, after the onset of cold weather. In the fall of 1925, when the writer made the investigation of fairs, referred to above, he was unable to locate any cases of hog flu on farms until the latter part of October. In 1926, no cases of flu were reported by members of the Eastern Iowa Veterinary Association during the months of September and October.

The season during which swine flu prevails in Iowa is a comparatively short one, the disease being confined chiefly to the late fall and early winter. As already pointed out, the disease seems to make its appearance with the onset of cold weather in the fall, varying somewhat according to whether the fall is an early or late one. Most of the cases seem to occur during the months of November and December, although scattered cases may be reported during January. The seasonal occurrence of the disease is quite well shown in the accompanying graph, based on data which were kindly furnished by the Eastern Iowa Veterinary Association. This association is a large and active one, with a membership of about one hundred and fifteen, and embraces forty-nine counties in the eastern part of the State. The association maintains a publicity committee, whose duty it is to collect and disseminate in the public press information relative to the prevalence of infectious diseases of live stock. All members of the association are supplied with printed, return post-cards upon which they make weekly reports of the various animal diseases prevailing in their respective localities. About half of the members make weekly reports in reply to the post-card questionnaires. Data relative to the occurrence of hog flu obtained in this manner should be quite trustworthy, as the veterinary profession in Iowa is now well acquainted with the disease.

During the period covered by the graph (i. e., from October 1, 1926, to February 1, 1927) the United States Weather Bureau at Des Moines, Iowa, reported the following weather conditions for Iowa: During October, 1926, the temperature for the State averaged nearly normal and there was no very cold weather. It will be noted that no cases of hog flu were reported in October. November, 1926, was a cold, disagreeable month, the coldest since 1911, and there was a decided deficiency in sunshine, especially in the extreme eastern portion of the State. The first really cold spell of the fall came between November 9 and Novem-

FIG. 1. Graph showing prevalence of hog flu in eastern Iowa, 1926-1927. (Based on data obtained from weekly news bulletins of the Eastern Iowa Veterinary Association.)

ber 11, when the temperature dropped to 11 degrees in the eastern part of the State and there was a heavy snowfall in this section on November 18. During December, 1926, the temperature averaged somewhat below normal and zero weather occurred in all parts of the State. January, 1927, was considered a mild winter month for Iowa, the mean temperature for the State averaging 3.2 degrees above the normal.

While the graph shown is for the flu season of 1926-1927, it would undoubtedly be quite typical of previous seasons with the exception that many more cases would have been reported by practicing veterinarians in the earlier years. The graph serves to bring out quite clearly the following points: (1) the appearance of the disease following the first cold weather in the fall, (2) the brief seasonal incidence of the disease, most of the cases occurring during November and December, (3) the rapid disappearance of the disease in the early part of January, due in large measure to the market shipment of the class of hogs that seem to be most susceptible to the disease, and (4) the small number of cases now reported by practicing veterinarians, twelve herds in one week being the maximum number reported by some fifty veterinarians, which goes to show that at present farmers seldom call the veterinarian. Formerly, when the disease was less well known, one veterinarian might have seen as many as twelve sick herds in a single day.

DINNER TO DR. VAN ES

On April 21, 1927, a dinner was tendered to Dr. L. Van Es, of the University of Nebraska, as an expression of the sincere appreciation and good will of the live stock and poultry interests and the medical and veterinary professions of Nebraska. The occasion took place in the Student Activities Building, Agricultural College, Lincoln, with Hon. Sam R. McKelire acting as toastmaster.

Addresses touching upon various phases of the science of animal pathology and hygiene were delivered by the following: Dean E. A. Burnett, Acting Chancellor, University of Nebraska; Dr. E. W. Rowe, president of the Lancaster County Medical Association; Prof. W. W. Burr, Assistant Director of Experiment Stations; Charles Graff, president of the Nebraska Improved Live Stock Association; and E. P. Browne, live stock breeder. Hon. C. H. Sloan spoke on "Man's Control of Live Stock Diseases."

BACTERIOLOGIC OBSERVATIONS ON PERIODIC OPHTHALMIA IN HORSES

Preliminary Report

By EDWARD C. ROSENOW, *Rochester, Minnesota*

Division of Experimental Bacteriology, The Mayo Foundation

Through the cooperation of Dr. F. Park Lewis, of Buffalo, N. Y., I have had opportunity to study the bacteriology of periodic ophthalmia in horses. The results have proved so interesting that a preliminary statement appears desirable at this time.

May 1, 1927, twenty-four of a herd of about seventy-five horses were found to have developed the disease on a farm near Buffalo. Of these, three developed their first attack in January, three in February, ten in March and eight in April. Four developed the disease during the last week in April. Only two had seemingly fully recovered. The condition of the affected eyes varied from mild lacrimation and congestion of the vessels with little or no turbidity of the fluid in the anterior chamber to marked clouding of the fluid, especially of the lower half, and marked circumcorneal congestion associated with or without opacity and vascularization of the cornea. Opacity of the lens resembling cataract, was found in only one instance. None of the animals had developed total blindness. The disease has been characterized by great fluctuations in severity, by a tendency to increasing involvement in successive exacerbations or attacks and by a tendency to spread to the unaffected eye, always without apparently affecting the general health of the animal. It affected equally the well-kept and more poorly nourished animals and the old and young alike, the age of the affected group varying from fifteen years to twelve days. The breed of polo ponies seemed more susceptible, for none of six draft horses kept on the farm under similar conditions had been affected. The disease was thought to be confined to this herd. However, on inquiry, several animals of neighboring farms also were found affected. The first attack in one of these animals began in February, about three months after having been pastured with the herd which became affected at about the same time. There was no evidence of spread of the disease by contact. It was not confined to animals

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kept in one stable, but occurred irregularly in widely separated individuals. Removal of eighteen of the animals to a somewhat higher altitude with a change of feed failed to check its progress. In agreement with the fact that these outbreaks are prone to occur in wet, damp, poorly draining soils is the fact that an unprecedented rainfall occurred last fall and early winter prior to the outbreak, making the pasture and enclosures extremely wet and muddy.

Bacteriologic Data

By means of animal inoculation or cultural methods, an organism having special affinity for the eye has been isolated from the lacrimal sac in nineteen of forty cultures made in sixteen of the affected animals. It has been isolated at least once in twelve of the affected animals, twice in four and three times in two. The inflammation of the eyes of the four animals that yielded negative results had either entirely disappeared or was receding at the time the swabbings were made. It has been isolated from the lacrimal sac in only two of twenty-eight normal horses of the herd affected and in no instance of twenty-three horses cultured remote from the outbreak. It has been isolated from the mucous membrane of the nose in two of six affected animals, from a sample of water containing yellowish pieces of scum or moss from the shaded side of the tub outdoors, to which the herd had access, and from the water contained in a tub within the stable supplying the drinking water for ten of the affected animals. Similar cultures from thirteen samples of water obtained from brooks, pools, hoof-prints, and wagon tracks in the pasture, failed to yield the bacillus in a single instance. It was isolated from one specimen of the oats and pea hay which was fed last winter when the epizootic began. It was not obtained in cultures from the feces of three nor from the winter coat of hair of one of the affected animals. It has been isolated from the fluid aspirated from the anterior chamber in four of six acutely affected eyes, and has been isolated in pure culture or in association chiefly with a large diplococcus from the fluid in the anterior chamber, and emulsions of the exudate covering the iris, and from the iris, uveal tract and limbus of an eye (removed under chloroform anesthesia for study) of a horse early in the third and worst attack.

Morphology and Cultural Characteristics

The suspected organism is a medium-size, rather slender, Gram-negative, motile rod with rounded ends. It grows rather

slowly aerobically on the common culture media. On plain or glucose agar and plain horse-blood agar it produces medium-size, glistening, translucent, non-adherent, yellow to orange colonies with smooth margins, and, on potato, a spreading, confluent, yellow growth. The yellow pigment formation is not so helpful for the isolation of the organism from mixed cultures as it would seem, for in some strains it does not develop until after forty-eight to seventy-two hours' incubation and may not appear at all where growth is heavy or where available oxygen is limited. In plain and dextrose broth it produces a moderate diffuse clouding, most dense in the upper layer and, in older cultures, a yellowish pellicle is formed. The pigment remains within the organism; fluorescence has not been observed. It produces no change in litmus milk, liquefies gelatin slowly, does not digest Loeffler's blood-serum, does not form indol, ferments sugars slightly without gas-production and reduces nitrates to nitrites. Its size and grouping vary considerably, depending upon whether grown in liquid or solid media. It usually stains diffusely although polar staining and clear areas somewhat resembling spores occur, in a few individual bacilli, especially in old cultures. On agar, the organism is quite uniform in size, ranging from 0.5 by 1.2 to 0.7 by 2.5 microns, and thread-formation is usually absent. In liquid cultures the length of individual rods varies considerably and short threads are sometimes seen. Flagella have not yet been demonstrated. Only a small proportion of the organisms are found actively motile, even in young cultures. It is not acid-fast. Young cultures are readily killed by heat and antiseptics, grows rather slowly at 35 to 37° C., and somewhat more quickly at room temperature; its optimal temperature for growth ranges between 25 and 30° C. According to Bergey's classification, the organism belongs to the genus Flavobacterium, but it does not conform in all particulars to any of the numerous varieties described as peculiar to this genus.

Great difficulty has been experienced in isolating and maintaining the organism in pure culture. It has happened repeatedly that fishings from well-isolated colonies on plates containing only characteristic colonies, of which smears revealed only Gram-negative bacilli and in which young sub-cultures proved to be pure, that Gram-positive or Gram-negative, large or moderate-size cocci or diplococci were found after the culture became old. This has been a puzzling problem and at present it is uncertain

whether we are dealing with inadvertent mixtures or mutations. The various forms found in these cultures, especially the large Gram-positive or Gram-negative diplococcus, resemble closely the organisms isolated commonly with the bacillus from swabbings of the lacrimal sac and from material obtained from within affected eyes.

Animal Inoculations

When injected in relatively large doses, the organism is virulent for the rabbit, guinea pig, white rat, and horse. The symptoms and death in each of these species, however, are due apparently chiefly to the presence of toxic material preformed in the broth or to that liberated from the organisms injected. The organism disappears quickly from the blood, after injection, in the animal that survives and is never found in large numbers, in blood and other tissues, in animals that succumb. It tends to localize in the anterior structures of the eye when injected intravenously.

The reaction in the animals injected in proper dosage is characteristic, especially in the rabbit. Soon after injection, in this animal, of from 2 to 5 cc of a broth culture or filtrate for each kilogram of body weight, the respirations become increased, weakness becomes manifest, often associated with fine tremors of muscles, especially on exertion, and coincidentally congestion appears in the vessels of the palpebral and ocular conjunctiva, of the iris and especially of circumcorneal vessels, and a variable degree of lacrimation occurs. If the animal recovers, the manifestations in the eye disappear. In one instance turbidity of the fluid in the anterior chamber and other evidence of infection have developed from intravenous injection. Placing the organism in the lacrimal sac, with and without injuring the cornea, has thus far not resulted in intrinsic infection of the eye. However, a condition strikingly like the specific disease has been produced in several horses and a series of rabbits by injecting into the anterior chamber a small amount of pure cultures and filtrates of old cultures. The disease has been induced in both eyes of one horse by injecting the fluid from the anterior chamber into one and the emulsion (in sodium chlorid solution) of the membraneous exudate covering the anterior surface of the iris into the other. The eye from which this material was obtained was removed on the fourth day of the third typical spontaneous attack. Marked clouding, especially in the lower half of the anterior chamber, occurred within twenty-four hours. The picture in both eyes

now, ten days since injection, is thus far typical of periodic ophthalmia. The bacillus was isolated from the material injected and from the fluid aspirated from the eye receiving the emulsion. Both of these strains have produced the characteristic reaction on intravenous injection in rabbits. Other organisms also isolated have shown little or no affinity for the eye and control injections of sterile broth are without effect. Filtrates of old cultures have been found extremely toxic, especially for the horse. Small amounts introduced into the anterior chamber cause marked clouding of the fluid, circumcorneal congestion, edema of the conjunctiva, and lacrimation lasting for a number of days.

Filtrates Are Toxic

It has been found that intravenous or subcutaneous injection of filtrates of exhausted cultures renders the eyes of rabbits and horses refractory to the effects of intraocular injections of the organism and filtrates, and in preliminary observations appears to lessen the inflammatory process in the eyes of affected horses. The filtrates are especially toxic for the horse. Two cubic centimeters injected intracutaneously and 2 cc subcutaneously sometimes produces, even in adult animals within from one to two hours, severe chilling, trembling of muscles, increased respiration and general illness in which the animals lie down and not infrequently manifest unmistakable circumcorneal congestion. The eyes of rabbits that have fully recovered following injection of the living organism or toxic filtrate and the eyes of horses that have fully recovered from the spontaneous disease, appear more refractory to injection of the filtrate than the eyes of normal animals, whereas the eyes of both of these species that still show inflammation are more susceptible than those of normal animals. The skin of the rabbit, guinea pig, goat, monkey, dog and white rat is highly immune to filtrates, even the undiluted filtrate giving little or no reaction, whereas the skin of the horse is extremely sensitive, dilutions as high as 1 to 1000 often giving decided reactions. Presumptive susceptibility tests with filtrates of this organism have been made in a series of normal horses and in affected animals. Great variations in reactions have been observed and a parallelism between the involvement of eye and degree of the skin reaction has been observed in some of the affected herd.

Rabbits and a horse are now being immunized in the hope of

developing an antitoxic curative serum. The fact that the organism appears to produce a specific toxin, measurable by intracutaneous injection, supports the hope that an antitoxin of high titre may be expected and which may perhaps be standardized by measuring its neutralizing power by intradermal injections of appropriate mixtures of toxin and antitoxin.

The results of the preliminary experiments indicate that the disease is probably hematogenous and not the result of a local infection through the conjunctival mucous membrane, although its common presence in the lacrimal sac in affected animals may be interpreted as indicating the latter route. Susceptibility of horses, as measured by intracutaneous injection of filtrates, varies within wide limits.

MANY CHANGES AMONG DETROIT VETERINARIANS

The veterinary profession in Detroit has seen many changes and realignments during recent months.

Dr. C. M. Hamilton (Ont. '26), who has been assisting Drs. E. E. and J. E. Patterson the past year, has purchased the practice of Dr. Charles H. Greenfield (Chi. '12). The latter is now assisting Drs. G. F. Ewalt and E. J. Burdick, who recently opened the Grand River Veterinary Hospital, at 10117 Grand River Avenue. Dr. Hamilton has Dr. H. S. C. MacDonald (Ont. '24) assisting him. Dr. Kenneth Ross (Ont. '27) has taken the place vacated by Dr. Hamilton at the Patterson Veterinary Hospital.

Dr. Paul V. Howard (Mich. '22) is again assisting Dr. A. L. Tow (Ind. '18). Dr. O. E. Markley (Gr. Rap. '17), formerly assistant to Dr. F. D. Egan (Ont. '23), is now in practice for himself, at 2620 John R. Street. Dr. George Cairns (Ont. '25), also formerly with Dr. Egan, has returned to Guelph, Ont. Dr. N. E. Christenberry (Ind. '23), formerly on the meat inspection force of the B. A. I., in Detroit, has resigned and opened the Blue Cross Small Animal Hospital, at 12463 Grand River Ave. Dr. S. R. Elkow (Ont. '23) has also opened a hospital at 13153 Hamilton Boulevard.

The morale of an organization is not built from the bottom up; it filters from the top, down.

—Peter B. Kyne.

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

A SUCCESSFUL TESTICULAR TRANSPLANT

By B. A. ZUPP, *Ames, Iowa.*

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The subject used in this expeirment was a mongrel male dog, about eight months old at the time of the first operation. He was a rather rangy individual, whose secondary sexual characteristics had not fully developed. He was kept under observation for several weeks to enable us to become familiar with his behavior and to accustom him to the environment.

On April 4, 1925, the right testicle and epididymis were removed and the testicle sectioned into four longitudinal slices. Only the two central slices were used. One slice of the testicle was immediately transplanted extraperitoneally and another was placed between the fibers of the restus abdominus muscle, anterior and lateral to the prepuce. The wound healed by first intention and soon after the dog developed priapism.

On May 21, the left testicle and epididymis were removed and the testicle transplanted on the opposite side, in a similar manner. The dog was observed daily and a condition of constant priapism developed.

On July 6, this dog was tried with a bitch in heat and he showed excessive desire and ability, but failed to complete coitus. This was possibly due to the fact that the bitch was considerably larger and that he lacked experience.

On July 20, 21 and 22, he completed coitus once each day and remained tied for from fifteen to twenty minutes.

On September 12 and 13 he was again tried on another bitch, but failed to complete coitus. He had sufficient desire but lacked ability. He made repeated attempts but with only a partial erection.

On November 4, another transplant was made, a testicle from another dog being used. One slice was placed in the rectus abdominus muscle between the prepuce and the fold of the flank, and a second slice was placed subcutaneously in the flank.

Following this operation the dog became lame and quite sore. Apparently the subcutaneous transplant underwent liquefaction necrosis. He made a rapid recovery, regained his ability and maintained his desire. In April, 1926, priapism had again developed. He was tried on a bitch going out of heat on June 3, but she would not permit coitus. He showed plenty of desire and ability.

On June 13, he was put with a bitch in heat and after many attempts he finally completed coitus and remained tied for fifteen minutes. On June 14, he tied the same bitch for fourteen minutes, on June 15 for thirteen minutes and on June 19 for thirteen minutes.

On February 24, 1927, he again completed coitus and remained tied for twenty minutes. This same bitch was then bred to a normal uncastrated dog of about the same size, who remained tied for nineteen minutes.

On April 13, 1927, he completed coitus twice the same afternoon and remained tied for twenty-one and thirteen minutes respectively. On April 13 he tied for nineteen minutes and on April 17 for twenty minutes. This bitch was then placed in the same kennel with a castrated dog who made the usual canine acquaintance and then lay down for a peaceful slumber.

This dog has been kept under extremely abnormal conditions, in a basement kennel, and his chief diet has consisted of dog biscuits. His masculine features have not developed and, seen from a distance, he appears rather feminine. However, he has retained his masculine habits. He will pick a fight with any dog and will win a large majority of the starts. It seems logical to assume now that some of his earlier failures to complete coitus were due to the fact that he was merely a pup and had never experienced coitus. The last coitus recorded was completed on the second attempt, while some of the first ones were not completed until after numerous attempts had been made.

Following each transplant, the dog developed priapism which at times was very pronounced and of long duration. Following the last transplant it was so marked that often the dog showed interference in gait and possible pain; the free end of the penis became dry, cyanotic and chapped. The priapism has gradually disappeared and at the present time his behavior is like that of a normal dog. In glandular transplantation there are three important factors to consider: first, that the transplant is immediate; second, that the sections of the gland to be trans-

planted are not too thick; and third, that the parenchymatous tissue of the gland is in direct apposition with the scarified tissue of the seat of implantation.

Summary The dog has been completely castrated since May 21, 1925, almost two years, and received his last transplant on November 4, 1925. He was an inexperienced pup to begin with, but now has the desire and ability to copulate equal to that of any normal uncastrated male.

This experiment is incomplete and it is planned to be continued over a period of years. A complete report will be published later.

RESPIRATORY DYSPNEA

By M. J. SEELY, *Lodi, California*

I was called to test an old Jersey cow which the owner suspected of being tuberculous. Upon slight exercise she did manifest a dyspnea which was suggestive of stenosis of some part of the respiratory tract. Upon ausculation of the heart no abnormality was detectable.

It was not a cardiac dyspnea, so the examination reverted to the head and throat regions. A leader was applied to the nose and a speculum adjusted. Upon examination of the laryngeal and pharyngeal regions and mouth, it was noted that the first upper molar was missing and the alveolus plugged tightly with feed.

A pair of forceps was brought into play and the mass removed. Some of the straws were six inches in length and were directed backwards and upwards through the maxillary sinus. The maxillary sinus was filled with food and inspissated pus.

After the cavity was thoroughly washed out, a metal catheter was passed through the alveolus into a fistulous tract, in a posterior-superior position, which revealed evidences of an old scar on the outside of the nasal bone. Upon inquiry, it was found that the old scar was formerly an abscess and had healed slowly. The nasal bone was trephined and it was found that the probe could be passed to the opening in the mouth.

The resulting low-grade inflammation had caused a swelling of the nasal sinus, thereby creating a diminution in the supply of inhaled air, resulting in respiratory dyspnea. The trephine opening, as well as the opening in the alveolus, were treated along surgical principles and the cow allowed no other feed but grass

and mashes. To date she has gained in flesh and has increased her supply of milk and upon exercise the respiratory distress is hardly evident.

TULAREMIA

By N. S. Mayo, *North Chicago, Ill.*

Within the past few years another disease has been added to the continually growing list of infections communicable from animals to man. It is probable that cases of tularemia occur in human subjects without being correctly diagnosed. Veterinarians should be posted on this disease.

Tularemia is caused by infection with *Bacterium tularense*, so name by McCoy, who described the germ as causing a "plague-like" disease of rabbits in Tulare County, California. This bacterium may be transmitted to man by the bites of various blood-sucking insects, fleas, ticks and bedbugs, and is also acquired by handling infected rabbits or by eating the meat of infected rabbits that has not been thoroughly cooked.

In man tularemia shows two forms, the glandular, which is more frequent, and the typhoid type. The period of incubation is from two to ten days. The disease is ushered in abruptly with profound prostration, generalized aching, and a chill, followed by a persistent high temperature. There is profuse sweating and frequently a slight cough, with conjunctivitis. In the glandular type the regional lymphatic glands become enlarged and tender. They may break, discharging a whitish, purulent material. Guinea pigs, inoculated with this pus, contract the disease. The complement-fixation test, with a special antigen, gives positive results. While a majority of human cases recover, the disease is prolonged and serious.

Veterinarians should watch for this disease as it seems to be appearing more frequently. Tularemia should not be confused with anthrax.

You Can Get Poultry Most Any Place Here

Two classified ads in the Alexandria (Ind.) *Tribune* would rather indicate that the poultry business is conducted as a side-line in that town.

FOR SALE—Nice fat hens at 25c pound. We will dress them free. Call electric light plant. Phone 44. Pearl Brobst & Sons.

FOR SALE—Young geese and ducks. Will dress them if you call early. Phone 83, or call Azimow's junk yard.

REVIEW

SPECIAL PATHOLOGY AND THERAPEUTICS OF THE DISEASES OF DOMESTIC ANIMALS. Franz Hutyra, Professor of Infectious Diseases, and Josef Marek, Professor of Special Pathology and Therapy, both of the Royal Veterinary College at Budapest. Third authorized American edition, translated from the sixth revised and enlarged German edition, and edited by John R. Mohler, A. M., V. M. D., D. Sc., Chief, Bureau of Animal Industry, U. S. Department of Agriculture, and Adolph Eichhorn, D. V. S., Director, Veterinary Department, Lederle Antitoxin Laboratories. Vol. I, xvi + 1091 pp., 259 figs. in the text and 19 plates; Vol. II, viii + 850 pp., 165 figs. in the text and 3 plates; Vol. III, vii + 835 pp., 185 figs. in the text and 6 plates. Alexander Eger, Chicago, 1926. Three volumes, silk cloth, $25.00.

This standard reference now makes its appearance in three volumes for the first time, previous editions having been in two volumes. The third English edition is a translation of the sixth German edition. During the ten years that elapsed between the publication of the second and third English editions, two new German editions were published. The latest, however, is really the most thoroughly revised edition of all and it contains material bearing upon important veterinary progress up to the year 1921. At first thought it might appear that this work is not up-to-date, but, when it is considered that these three books really contain a digest of our scientific knowledge relating to animal diseases all over the world, it becomes readily apparent how stupendous a task the careful preparation of such a voluminous and comprehensive publication really is.

American veterinarians will be disappointed if they expect to find in a work of this kind what might be termed "recent developments" in veterinary science in this country. It should be kept in mind, however, that articles and reports appearing in our own scientific publications must first be translated and the new material then incorporated in a new German edition, which, in turn, must be translated into English. This is a time-consuming process and explains why this new American edition contains very little material more recent than 1921. It is pleasing to note the increasing number of names of American investigators that are finding their way into this work.

Volume I, prepared by Hutyra, is devoted exclusively to infectious diseases. These are divided into six groups: (I) Acute general infectious diseases. (II) Acute exanthematous diseases. (III) Acute infectious diseases with localization in certain organs. (IV) Infectious diseases with special involvement of the nervous system. (V) Chronic infectious diseases. (VI) Infectious diseases produced by protozoa.

New material in the first volume includes a chapter on paratyphoid. Under this head are considered those affections in horses, cattle, sheep and pigs believed to be caused by bacteria of the paratyphus group. The importance of this particular infection is just coming to be recognized and it is timely that the author should devote approximately eighteen pages to the discussion of our present knowledge of the disease.

The chapter on Texas fever is new and this disease is recognized as a distinct form of piroplasmosis. Most of the material on this disease is based on the work that has been done by American investigators.

The chapter on malignant edema reflects the vast amount of work that has been done on this disease during recent years. The same may be said of ulcerative and epizootic lymphangitis. Likewise the material dealing with infectious abortion has been thoroughly covered.

Volume II, prepared by Marek, contains a number of the organic diseases. Much new material is found in this volume, including acetonemia of cattle, dochmiasis of silver fox, intestinal coccidiosis of swine.

In this volume the material is divided into three major sections: diseases of the digestive organs; diseases of the respiratory organs; and diseases of the circulatory organs. Each of these sections is appropriately subdivided into sections covering the diseases of the individual organs. There are sixteen such subdivisions.

Diseases of the stomach and intestines take up almost one-half of this volume. This is accounted for, in part, by the fact that gastro-intestinal parasites of all species are considered here.

Volume III, also prepared by Marek, contains much new material on such diseases as lamziekte, encephalitis lethargica, louping-ill of sheep, polyneuritis, etc. Many of the chapters in this volume have been entirely or partly revised and elaborated.

The third volume is really a continuation of the second and it covers the following: diseases of the urinary organs; diseases of

the blood and blood-producing organs; diseases of the spleen; diseases of metabolism; diseases of the nervous system; diseases of the organs of locomotion; and diseases of the skin. Approximately 261 pages are devoted to diseases of the skin, both parasitic and non-parasitic. It is noted that infectious anemia of horses is discussed in the third volume, rather than in volume I with the other infectious diseases.

The authors have attempted to give due consideration to those diseases which occur outside of Europe, according to their importance. Emphasis has been placed on etiology and diagnosis, as it is recognized that intelligent handling of any of these diseases is largely dependent upon these factors. In addition to their own observations, the authors have recorded the studies and findings of many other investigators, through a careful examination of the literature. To a certain extent this phase of the work has been delayed, due to the interruption of the publication of reports in a number of foreign countries occasioned by the war.

The text is well illustrated, over 100 half-tone illustrations having been added and the number of full-page plates (many in colors) increased from twenty-one to twenty-eight. Most of these are very fine illustrations of the pathological conditions represented. Each volume is separately indexed. The publisher has spared no expense in making this English edition available to American veterinarians. It is worthy of a place in the library of every member of the profession. It is almost inconceivable that any veterinarian could get along without it, no matter in what branch of the profession he may be engaged.

BUREAU TRANSFERS

Dr. Peter A. Franzmann (Cin. '09), from Davenport, Iowa, to Seattle, Wash., on meat inspection.

Dr. Joseph Hannon (McK. '10), from Chicago, Ill., to Davenport, Iowa, in charge of meat inspection.

Dr. Cloyd M. Riley (McK. '09), from Tacoma, Wash., to Portland, Ore., on meat inspection.

Dr. Lynce C. Bowling (Iowa '20), from Chicago. Ill., to Tacoma, Wash., on meat inspection.

Dr. Chas. A. Hazzard (K. S. A. C. '12), from Chicago, Ill., to Houlton, Me., on meat inspection.

Dr. James R. Donnelly (Ont. '07-McK. '08), from Pittsburgh, Pa., to Pittston, Pa., in charge of meat inspection.

Dr. Harry D. Larzelere (O. S. U. '09), from hog cholera control to tuberculosis eradication, Madison, Wis.

Dr. Mark Heusinkveld (Chi. '18), from Chicago, Ill., to Sioux City, Iowa, on meat inspection.

Dr. Wm. J. Selkin, from Jersey City, N. J., to New York, N. Y., on meat inspection.

ABSTRACT

BLACK-QUARTER IN SOUTH AFRICA: WITH SPECIAL REFERENCE TO IMPROVED METHODS OF INOCULATION. P. R. Viljoen and J. B. Scheuber. 11th and 12th Reports of the Director of Veterinary Education and Research, Union of South Africa, Department of Agriculture, Pretoria, 1926, pp. 499-570.

The authors discuss the occurrence of blackleg in South Africa and its control. They find that blackleg artificial aggressin (filtrate) is the best method of controlling the disease. Experiments on powder vaccines tested on sheep show that these vaccines are practically of no value, the best vaccines protecting against only one m. l. d. of virus and in many cases causing losses in vaccinated animals. A natural aggressin also was produced in calves, which protected animals against 2 m.l.d. of virus. The disadvantages of this method of protection are stated (p. 531).

The main disadvantages connected with this method of vaccination are the suffering inflicted on animals, its costliness, and the extra work entailed in its preparation. The points referred to hardly require any further explanation; calves are used in the manufacture of the vaccine, and it is quite common for them to go through an acute attack of black-quarter, lasting for many hours, during which they must suffer terrible agony; the period of suffering can not be shortened and they must be allowed to die a natural death, otherwise the production of aggressin is interfered with considerably. According to Zschokke, an average of 8 litres of aggressin could be obtained from a single calf, but in our experience the quantity was much smaller; this alone makes the vaccine expensive. Even the simplest technic that could be employed would involve a great deal of labor, and this further increases the cost of production. If this aggressin were greatly superior to any other form of black-quarter vaccine, the extra cost of production would be justifiable, but the authors are of the opinion that an equally efficient vaccine could be produced in a much simpler and cheaper manner. The vaccine referred to is the so-called artificial aggressin. The summary and conclusions of the authors follow:

(1) Black-quarter has been known to exist in South Africa ever since the first veterinarians came to this country. At the present time it is widespread, occurring as an enzootic in many parts of the country, and being particularly prevalent in low-lying areas.

(2) Natural infection occurs in young cattle and, to a smaller extent, in sheep. In the latter animals, the disease often makes its appearance on a large scale, infection taking place through wounds in the skin.

(3) It is not a scheduled disease under the Stock Diseases Act, the State departments concerned acting only in an advisory capacity. Control is left to the individual stock-owners, who are given full advice concerning the best methods of prevention that are known to science. Losses from the disease can be avoided by timely inoculation of susceptible stock with a reliable vaccine.

(4) Bacterial vaccines are based on the principle that the organisms themselves are largely responsible for the immunizing value of vaccine. This has now been proved to be wrong, the "washed" organisms having neither pathogenic properties not immunizing value. To render such a vaccine safe for use, the number of organisms contained in it must always be kept at a minimum, and in doing this the immunizing value (aggressin-content) is often greatly reduced. Attempts on a big scale to find a method of preparation that would give a vaccine which could be considered both safe and efficient have ended in failure.

(5) Efficient anti-sera can be obtained easily by hyperimmunizing sheep with either virulent cultures or germ-free filtrates. Such sera may be used in cases where it is desired to obtain an immediate protection or in conjunction with virulent cultures or bacterial vaccines. Under the conditions prevailing in this country, there has so far been no need to resort to the use of anti-sera.

(6) The writers have never detected any sign of toxin-formation in their black-quarter cultures.

(7) The immunizing principle in black-quarter is a bacterial product which is formed during the active multiplication of the causal organism, either in the animal tissues or in suitable artificial media. Owing to the aggressive character which it confers on the organism, this substance has been termed aggressin; when it is produced in the animal body it is called "natural aggressin," and when it is formed in artificial cultures it is usually referred to as "artificial aggressin."

The writers have not been able to detect any real differences between these two forms of aggressin and are forced to regard them as identical. Apparently the one produced in the animal body is formed under ideal conditions, and is therefore present with more certainty and in greater abundance; the other is produced artificially, and the degree or intensity of its production must, and undoubtedly does, depend on the nearness to which one is able to approach the artificial and natural conditions of bacterial growth. The efficiency of an artificial aggressin as an immunizing substance depends, therefore, on the perfection to which the artificial cultivation of the organisms can be brought.

The following properties possessed by aggressins are of considerable importance:

(a) They are absolutely non-toxic and germ-free.

(b) They have excellent keeping properties; natural aggressin preserved in 60 per cent glycerin has kept for two and a quarter years, and artificial aggressin for over a year, without their immunizing properties being lost; natural aggressin resists heating at 80°C. for half an hour; artificial aggressin, 95°C. for half an hour, and direct sunlight for 100 hours.

(c) They confer a strong, lasting immunity; vaccinated sheep still showed immunity twelve months after inoculation. Artificial aggressin has a great advantage over natural aggressin, in that it can be prepared much more cheaply and simply.

(8) A well-marked variation in the virulence and immunizing value of black-quarter strains from different localities has been established. The variation in virulence is not of great practical importance, since with an efficient vaccine it is possible to immunize animals against all other strains.

(9) A good immunizing vaccine (filtrate) against malignant edema can be prepared by following the technic employed by us for the preparation of black-quarter filtrate.

(10) Vaccination against black-quarter affords no protection against infection with malignant edema and vice versa.

(11) A mixed vaccine for use against both diseases can be obtained by growing the respective organisms separately and then mixing the two filtrates in equal proportions.

(12) During the last few years, black-quarter filtrates (artificial aggressins) have been used extensively in South Africa, the annual issues being wel lover a quarter million doses; for 1924 the figure was 268,040 doses. The results obtained have been extremely satisfactory, so much so that complaints in regard to the efficacy of the vaccine are practically unknown. Experience has shown that a black-quarter vaccine may be considered satisfactory as long as animal tests show it to be efficient against at least one minimum lethal dose of virus. This was the accepted standard with the first artificial aggressins that were issued, and we have on record cases where, with this vaccine, farmers were able to stop all mortality from black-quarter on extremely badly infected farms; in fact, in some of these cases repeated vaccination with other preparations had been tried unsuccessfully. We now use vaccines of a higher standard, protection being required against at least two or three minimum lethal doses of virus. Several thousand doses of natural aggressin have been used also with great success; this vaccine has not been used more extensively simply because the more easily prepared and cheaper artificial aggressin has given every satisfaction.

A comprehensive bibliography of eighty titles is appended.

J. P. S.

PUBLICATIONS RECEIVED

American Association for the Advancement of Science. Booklet of Information on the Organization and Work of the Association. Washington, D. C., 1926. pp. 8.

Radiometric Measurements on the Carbon Arc and Other Light Sources Used in Phototherapy. W. W. Coblentz, M. J. Dorcas and C. W. Hughes. (Scientific Papers of the Bureau of Standards, 539. Department of Commerce, Washington, D. C., 1926. pp. 535-562.)

Ontario Veterinary College, 1926, Report of the. Ontario Department of Agriculture, Toronto, Ont., 1927. pp. 69. Illustrated.

University of Pennsylvania Bulletin, School of Veterinary Medicine, Announcement for 1927-1928. Philadelphia, Pa., 1927. pp. 48.

Basal Metabolism Determinations on Dogs. (Research Bul. 5, Parke, Davis & Co., Detroit, Mich., April, 1927. pp. 13. Illustrated.)

Tenessee, Biennial Report of the Department of Agriculture for 1925-1926, State of. Nashville, Tenn., March 1, 1927. pp. 68. Illustrated.

The Moore Important Poultry Diseases. L. Van Es and H. M. Martin. (Bul. 195, Univ. of Nebr., Coll. of Agr. Exp. Sta., Lincoln, Nebr., October, 1923. pp. 79. Illustrated.)

A Study of the Tumor Incidence in the Lower Animals. W. H. Feldman, Boston Mass. Reprint from *American Journal of Pathology*, ii (1926), 6. pp. 545-556.

Studies on the *Ascaris Lumbricoides*. H. M. Martin. (Res. Bul. 37, Univ. of Nebr., Coll. of Agr. Exp. Sta., Lincoln, Nebr., December, 1926. pp. 78. Illustrated.)

Feeding for Egg Production. L. E. Card. (Cir. 275, Univ. of Ill., Agr. Coll. & Exp. Sta., Urbana, Ill , November, 1923. pp. 12. Illustrated.)

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

Lt. Colonel Walter R. Pick, now on duty at Fort Omaha, Nebraska, will report by letter to the district engineer, United States Army, 251 Federal Building, Rock Island, Illinois, for duty as meat inspector for the Corps of Engineers at South Omaha, Nebraska, in addition to his present duties.

Major Ralph M. Buffington is relieved from assignment and duty with the Second Medical Regiment, Fort Sam Houston, Texas, effective on or about July 5, 1927, and will then proceed to Fort Myer, Virginia, and report in person to the commanding officer for duty.

Reserve Corps

Promotions

Carter, Thomas Byron, 709 E. 29th St., N., Portland, Oregon, to the grade of captain.

New Acceptances

Hinson, Lonnie Jefferson, 1st Lieut., Greenwood, Miss.

Powell, Thomas Maurice, 2nd Lieut., c/o Zoller Packing Co., Spring Garden Ave., Pittsburgh, Pa.

Separations

Ackerman, Norman Walker, 2nd Lieut., Resignation accepted, 4-2-27.

Davis, Howard Burtis, 1st Lieut. O. R. C. status terminated because of National Guard status.

Leavitt, Charles Henry, 1st Lieut. Resigned.

THE GIRAFFE SPEECHLESS

In a recent issue of *Farm and Fireside*, Dr. W. Reid Blair, director of the New York Zoological Park, had a very interesting article, in which he described some of the peculiarities of a few of the wild animals in his charge. Many newspaper comments on the article have appeared. For example, the *Evening Bulletin* (Philadelphia) says that the article contains enough material for an evening's amusement at the new popular pastime of questions and answers, and then quotes Dr. Blair as follows:

> The giraffe is the only mammal which has no voice. The penguin is one bird that never builds nests. The egg is carried on top of the webbed foot of the mother bird and warmed with a roll of body fat. When the female wants a rest, her mate sidles up close and with a neat move the egg is passed over to his foot for him to keep warm.

C. C. B., the columnist of the *Free Press* (Detroit), was evidently impressed by the fact that the giraffe has no voice. He remarked: "But that's all right. Can you imagine anything more tiresome than craning your neck to hear a speech from a giraffe?"

Well, C. C. B., we have heard some humans that were awfully tiresome speakers.

COMMUNICATIONS

BOOKLET STOPS ARGUMENTS

To the Editor:

Enclosed find one dollar for which please send me one hundred of the little booklets, "Nothing to Sell but His Services." They head off arguments and settle many disputes. I used a hundred of them before.

Fred W. Lange.

Salem, Oregon, April 26, 1927.

SOURCE OF INFORMATION

To the Editor:

You have made the Journal a great source of information on things that very much interest me. The printing of the report of the U. S. Live Stock Sanitary Association in the March number was a good idea, and I was pleased to have the opportunity of reading it. I wish you the best of good luck for the coming year, and that the Journal will continue to flourish.

George Townsend.

New Glasgow, N. S., April 23, 1927.

FROM THE PHILIPPINES

To the Editor:

As requested in your editorial in the March number of the Journal, I am writing to express my appreciation for the publication of the proceedings of the thirtieth annual meeting of the United States Live Stock Sanitary Association, which I think carries very instructive information to all interested in live stock. I am therefore strong for the idea in publishing the report annually.

V. Buencamino.

Manila, P. I., April 25, 1927.

STATE BOARD EXAMINATION

Iowa Veterinary Examining Board. State House, Des Moines, Iowa. July 1-2, 1927. Dr. P. Malcolm, Secretary, State House, Des Moines, Iowa.

MISCELLANEOUS

GOOD SERUM AND THE VETERINARIANS

A recent bulletin from the U. S. Department of Agriculture reports a shortage of veterinarians and stresses the fact that but few young men in our colleges are turning to that profession.

It is a known fact that so few have become the veterinary students that many of our good veterinary colleges, which in the past were big and flourishing institutions, have closed their doors. It has happened to such a degree that complete veterinary courses are offered in only a few institutions in the entire country. This condition is of the gravest concern and its remedy of the most vital importance to every farmer in the United States, especially so to every one of us interested in the great live stock industry. It is, however, a situation entirely to be expected and no one has brought it about but the very farmers and live stock men who are most dependent upon skilled veterinary service.

With the changed present-day agricultural conditions and the consequent decrease in the general horse and cattle practice of the veterinarian, especially here in the corn and hog belt, was he almost forced to become a hog specialist. Swine treatment became the backbone of his practice, his very bread and butter; of that hog practice, cholera vaccination and immunization made up a considerable portion. We hog men should have welcomed this specialization in our line—should have developed and encouraged it—but what did we do?

Just as soon as the basic underlying principles of vaccination and cholera immunization became a little better known, hog breeders and pork producers, in increasing numbers each year, began to treat their own hogs with serum purchased directly or had them treated by their county agent. It is true that during the past few years, due to the stress of the times, some farmers and breeders were actually forced to dispense with veterinary service. It was no fault of their own and now that the economic condition of the hog business has again adjusted itself they will promptly discard the practice.

We have no quarrel with the county agent who is sincerely endeavoring to give his community real service. Quite to the contrary, we believe in the county agent and are with him and for him in every conceivable way. When he undertakes to vaccinate

hogs, in our opinion, he has stepped out of his rightful province and fails absolutely in giving that service which he and his members may wrongly believe he is giving. Again in our opinion, he is usurping the field of an important man in our live stock scheme. He is taking away the recognized work and means of livelihood of that man whose services he cannot even begin to replace when something else is needed other than vaccination.

A real honest-to-goodness veterinarian, well versed and up on his toes, who positively knows his business, aside from being absolutely vital to his community, is an added valuable asset to the live stock improvement of that community. Yet some of our farmers want to take away a big slice of his living and still expect him to stay there to come at any hour of the day or night that Dobbin may show signs of an impending case of tetanus, when the spotted cow rolls up her eyes in the throes of milk fever or when something springs up in the hog lot that can't be recognized.

In the cholera immunization of hogs, whoever does the work is handling actual cholera germs in their most virulent form and it is our own firm conviction that this is not a layman's job. If a veterinarian does the job, he will and should make a reasonable charge for his work, but it is worth that charge for his experienced watch over the herd, immediately after vaccination, for possible "breaks" alone. A competent veterinarian can run through a bunch of hogs in a couple of hours that it would take the inexperienced neighbor help a day or more to dispose of. Farmers who have tried both methods have repeatedly told us that there is no real saving in home vaccinating talent; that extra help, extra meals for that help and less positively dependable results more than make up for the apparent difference. Known to us personally are a number of efficient veterinarians who vaccinate thousands of hogs each season who haven't had a vaccination abscess develop in years and that does not come with home vaccination or home vaccination methods. All of these things made the veterinarian's charges for his services a regular and justifiable expense of the business which we should gladly assume in light of direct benefit and value received. Isolated cases of unreasonable vaccination charges on the part of unscrupulous veterinarians will readily adjust themselves with proper handling.

To place the veterinarian in the position of attempting to compete with the county agent and our own services in vaccination is absurd, the only possible result being the increasing of

his temptations to use a cheaper and inferior serum and to decrease the dosage. Such a practice would be ruinous to our own business.

Therein lies another of our pet hobbies. There is no possible excuse or justification for anything but the very best of serum. The manufacture of an unquestionable grade of potent serum and virus is both a costly and highly technical art. So very much is at stake in what comes out of a serum bottle that we cannot afford to intrust our whole success in our chosen business to any but a serum manufacturer in whose skill and integrity we have the utmost confidence. Fortunately there are a number of that kind. They must of necessity charge for their wares, a price consistent with the production cost and value of their product. Suspiciously cheap serum prices very frequently have led back to "fly-by-night" serum manufacturers, who periodically have their licenses revoked for putting out impotent serum. Just one experience with that sort is a thousand times more costly than the extra expense over a lifetime for a product of recognized high quality, competently administered by a veterinarian.

The cholera epidemic of last year again temporarily re-taught us the costly lesson of the value of keeping our herds immunized at all times. It should not stop there. See to it that a high grade, reputable serum of established and unquestionable value is used and let it be administered by a good veterinarian. Know the veterinarian and know just what serum it is that comes out of the bottles that he uses.

There will then be no further alarm over the failure of the young men going into the veterinary profession in ample numbers to supply our needs—which are both real and vital.

—*The American Herdsman.*

DR. BEMIS TO PENNSYLVANIA

Dr. H. E. Bemis, who, on April 18, 1927, was elected to succeed the late Dr. John W. Adams as Professor of Surgery and Obstetrics in the School of Veterinary Medicine, University of Pennsylvania, is one of the leaders of the veterinary profession in this country and the outstanding man in his special line of work. Dr. Bemis, who is a native of Kansas, after his graduation from Northwestern Academy, attended the School of Arts and Science in Northwestern University. Later he entered the Iowa State College and was graduated from that institution with the

degree of Doctor of Veterinary Medicine in 1908. In the fall of that year he returned to the institution and accepted an appointment as Instructor in Surgery and Obstetrics and was subsequently promoted to Assistant Professor, then to Associate Professor, and finally, in 1915, to Professor. In the same year he was appointed Vice-Dean of the Veterinary School.

Dr. Bemis has been an active research worker and has made a number of valuable contributions to veterinary medicine. His work in connection with the development of methods of local anesthesia to replace general anesthesia in certain major operations has proved of great value in veterinary surgery, while his studies of diseases of the genital organs of the dairy cow have been attended with interesting developments. For the last ten years Dr. Bemis has been much in demand to conduct surgical and clinical programs at veterinary conferences in the central western states.

During the World War he was an officer in the Veterinary Corps of the U. S. Army and served with distinction in France. Among his more important assignments were Division Veterinarian, 89th Division; Commanding Officer, Veterinary Hospital No. 3; Assistant to Chief Veterinarian, A. E. F.; and Chief Veterinarian, Third Army, A. E. F. He entered the service with the rank of Major and was discharged with the rank of Lieutenant-Colonel.

At the completion of the present session at the Iowa State College, Dr. Bemis plans to go abroad for study and will return in time to begin his work at the University of Pennsylvania at the opening of the next session.

G. A. Dick,
Supervisor, Veterinary Extension,
University of Pennsylvania

DEAN STANGE FEELS LOSS OF DR. BEMIS

Dr. C. H. Stange, Dean of the Veterinary Division, Iowa State College, made the following comment on the resignation of Dr. Bemis:

> His going is a great loss to Iowa and to the College. In his profession, Dr. Bemis is recognized as one of the great surgeons of the country. We congratulate the University of Pennsylvania on securing his services.
>
> I deem his resignation a distinct personal loss for I have been associated with him actively for nineteen years. It is too bad that Iowa does not feel that it can afford to retain men of such exceptional ability. However, we are glad that Iowa State College is capable of producing such men.

COMMENCEMENTS

ONTARIO VETERINARY COLLEGE

The spring examinations of the Ontario Veterinary College, Guelph, Ontario, were completed on April 28, and graduation exercises were held the following day.

The degree of Bachelor of Veterinary Science was conferred at a special Convocation of the University of Toronto on ten graduates as follows: John C. Bankier, Leo F. Barthelme, R. Paul Dingman, H. P. Hamilton, John E. Howard, Kenneth Ross, Charles A. Skuce, Carson M. Steen, Ralph V. Westerberg, and Francis M. Wheatley.

Honors were awarded to members of the graduating class as follows:

General Proficiency

First Prize—J. C. Bankier, of Glasgow, Scotland.
Second Prize—L. F. Barthelme, of Labette, Kan.
R. V. Westerberg, of Simsbury, Conn.
Third Prize—R. P. Dingman, of Prophetstown, Ill.

Bacteriology

Special Prize—J. C. Bankier, of Glasgow, Scotland.

Helen Duncan McGilvray Honorarium

L. F. Barthelme and J. C. Bankier.

K. S. A. C. PRIZES

The fourth annual Recognition Day program of the Kansas State Agricultural College took place in the College Auditorium, May 5, 1927. At this time, all winners of student prizes in the College received their prizes and awards. The names of the winners of the prizes offered the students of the Division of Veterinary Medicine are as follows:

General Proficiency

(Prizes offered by Dr. E. A. Schmoker (K.S.A.C.'17), of Monroe, Wash.)
First Prize ($10.00)—Earl Francis Graves.
Second Prize ($5.00)—Elmore Franklin Sanders.

Therapeutics

(Prizes offered by the Jensen-Salsbery Laboratories, of Kansas City)
First Prize ($10.00)—Ray Lewis McConnell.
Second Prize ($5.00)—Floyd Eugene Carroll.

Pathology

(Prize of $7.50 offered by the Veterinary Faculty of the College)
Earl Francis Graves.

Physiology

(Prize of $7.50 offered by the Veterinary Faculty of the College)
Lawrence O. Mott.

AMERICAN VETERINARY MEDICAL ASSOCIATION

Report of the Tenth Annual Meeting of the Women's Auxiliary to the A. V. M. A., Lexington, Kentucky, August, 17, 1926

Opened with the address of welcome by Mrs. Thomas P. Cooper, of Lexington, Kentucky, in which the visiting ladies were extended the far-famed hospitality of Kentucky. The response was given by Mrs. S. E. Hershey, of Charleston, West Virginia, who assured the Kentucky ladies that the visitors were delighted with Lexington and had been looking forward with pleasure to their visit to the Blue Grass region with its world-wide reputation for hospitality and its "Thorobreds."

Mrs. C. E. Cotton, of Minneapolis, president of the Auxiliary, in her address, emphasized the fact that the men of the A. V. M. A. were not yet awake to the real purpose and importance of the Women's Auxiliary. She stated that in her opinion they looked upon it as primarily a social organization, whereas its real function is usefulness, and expressed the hope that the next few years would see a decided change in the general attitude toward the organization.

Mrs. C. H. Stange, of Ames, Iowa, presented a symposium upon "The Student Loan Fund." "The Past" was covered by Mrs. A. T. Kinsley, of Kansas City, who stated that the Fund was established originally to give help to needy families of veterinarians during the war, but later was converted into a fund for helping worthy veterinary students. Up to August 1, 1926, $2,425.00 had been loaned to nine students. "The Present" was presented by Mrs. H. P. Hoskins, of Detroit. She stated that the organization was in excellent condition financially, and that in October, 1926, it would be in a position to repay the entire loan of $1,200.00 made from the Salmon Memorial Fund through the Executive Board of the A. V. M. A. "The Future" was discussed by Mrs. C. H. Stange, of Ames, Iowa, who made two suggestions:

1. *Publicity.* This is a necessity for the progress of any organization.

2. *Increased Membership.* This is a natural result of proper publicity. Let it be remembered that "Today is better than yesterday and progress is the law of life."

The entire meeting was better attended, showed greater enthusiasm and in every way was superior to any previous one. Here's hoping that the organization may perpetuate this spirit and develop as it should the opportunities which are before it.

MRS. S. E. HERSHEY, *Secretary.*

DOCTOR STEEL LEAVES IOWA

Dr. E. R. Steel, the energetic secretary of the Iowa Veterinary Association and of the Missouri Valley Veterinary Association, has left Grundy Center, Iowa, and returned to Kansas City, his former home. He is erecting a small animal hospital on one of the main thoroughfares of Kansas City, near the city limits. Dr. Steel has resigned the secretaryship of the Iowa Veterinary

DR. E. R. STEEL

Association and the Executive Committee has filled the vacancy by appointing Dr. C. J. Scott, of Knoxville, for the unexpired term. Dr. Steel will continue as secretary of the Missouri Valley Veterinary Association, a position which he has filled with conspicuous success for four years.

RECIPROCITY AGREEMENT

The veterinary examining boards of the states of Ohio and Michigan recently entered into a reciprocity agreement for the exchange of licenses to practice in the respective states.

ASSOCIATION MEETINGS

VETERINARY MEDICAL ASSOCIATION OF NEW JERSEY

The forty-third annual meeting of the Veterinary Medical Association of New Jersey was held in conjunction with the fourth annual conference for veterinarians at the College of Agriculture, New Brunswick, January 25-26, 1927. More than sixty veterinarians were in attendance, including a number from neighboring states.

Tuberculosis and contagious abortion of cattle were the principal problems considered. In discussing tuberculosis, Dr. J. A. Kiernan, chief of the Tuberculosis Eradication Division, U. S. Department of Agriculture, emphasized the importance of area testing in order to eradicate the disease most economically and efficiently.

Dr. E. C. Schroeder, superintendent of the B. A. I. Experiment Station at Bethesda, Md., expressed the belief that the eradication of bovine tuberculosis has even greater significance to human health than to the economic welfare of the dairymen.

Dr. L. F. Rettger, professor of bacteriology at Yale University, laid considerable stress on the necessity of testing cattle to eliminate those animals infected with contagious abortion. He warmly commended the New Jersey Legislature for providing the means to control this disease.

The progress of veterinary medicine was reviewed by Dr. David S. White, dean of the College of Veterinary Medicine, Ohio State University. He insisted that the veterinary profession needs frequent conferences and that they should be conducted largely on the plan of graduate instruction. Dr. Howard J. Milks, of the New York State Veterinary College at Cornell University, talked on canine diseases.

The life and work of the late Sir Stewart Stockman, of England, was reviewed by W. C. Thompson, professor of poultry husbandry at Rutgers. Professor Thompson made it clear that veterinary medicine owes considerable to Sir Stewart. Dr. F. R. Beaudette, poultry pathologist at the Experiment Station, presented facts to show the effectiveness of recent measures for controlling internal parasites in poultry. In this talk it was brought out that he has had great success in raising chicks in confinement.

The recent outbreak of vesicular stomatitis in Sussex County and the methods used for stamping it out were discussed by J. H. McNeill, chief of the Bureau of Animal Industry, State Department of Agriculture. Dr. McNeil was elected president of the Association for the coming year.

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held in the Academy of Medicine Building, 5th Avenue and 103rd Street, New York City, Wednesday evening, April 6, 1927, at 8:30 p. m.

President C. G. Rohrer presided. There were thirty-eight in attendance. The minutes of the March meeting were read and approved.

Dr. Maurice C. Hall, Chief, Zoological Division, Bureau of Animal Industry, Washington, D. C., gave us a very instructive and interesting paper, illustrating by lantern-slides the live stock conditions in Central America, with reference to parasites there and in the United States. He explained and showed by slides the abattoirs for slaughtering cattle and hogs; also the methods for the inspection of meat, which are very different from those used in this country. He mentioned that the inspection is done by an officer of the law and not a veterinarian; also that the animal is slaughtered and most of the meat is used the same day.

In comparing the parasites found in Central America with those in the United States, Dr. Hall stated that they were about the same, although some are much more prevalent. He indicated that the prevalence of certain types of worms and the absence of their intermediate hosts suggested a field for more study to clear up their life cycles. He also pointed out the great differences in the parasites found in animals, due to the different climatic conditions, dry or wet, range or quartered. They found many human subjects infested with pinworms and hookworms.

Dr. Hall gave the members a very good description of the Canal Zone, with reference to geography, inhabitants, manner of living and government. He explained the conditions under which the natives are compelled to work, in their rather makeshift laboratories, and the reluctance of the human subject to undergo treatment.

Dr. Hall made good use of his spare time by writing poetry, which he read to us. For a research man located in Washington, he is quite a poet. He read one poem, depicting the Spanish girl, which showed his judging ability as to the human female sex. He, too, thinks it a good thing for one to have a hobby (such as writing poems).

The discussion was opened by Dr. C. V. Noback, who reported his experiences in Bolivia, South America. A rising vote of thanks was extended Dr. Hall for his contribution to the program.

Dr. Dell C. Bassett, of Hempstead, L. I., was elected to membership in the Association. The resignation of Dr. Robert Dickson, of Little Silver, N. J., was accepted and on motion by Dr. R. S. MacKellar, duly seconded, the by-laws of the Association were suspended and Dr. Dickson was elected an honorary member.

Dr. R. W. Gannett moved, and it was regularly seconded, that the resignation of Dr. Geo. W. Little be laid on the table and a committee be appointed to call on him to see if he would not remain with the Association. The president appointed Drs. J. E. Crawford and R. W. Gannett.

Under new business, Dr. C. S. Chase suggested that the Association consider the finding of a different meeting-place, since the present Academy of Medicine is out of the way for a large number of members. The president and secretary were asked to investigate.

Dr. O. E. McKim reported articles appearing in the New York *Tribune*, written by Frank Dole, in reference to rabies and inoculations against same. Discussion was entered into by Drs. Ticehurst, Gannett, Goubeau, Miller, Amling, Eichhorn, Crawford, Higgins and Appleberg.

Dr. MacKellar announced that the New York State Veterinary Medical Society would meet in Watertown, N. Y., June 28-29, 1927.

No further business appearing, the meeting adjourned.

C. P. Zepp, *Secretary.*

NORTHEASTERN PENNSYLVANIA VETERINARY MEDICAL CLUB

A meeting of the Northeastern Pennsylvania Veterinary Medical Club was held at the Terminal Hotel, Wilkes-Barre, April 21, 1927, with an attendance of fifteen. Dr. B. M. Under-

hill, of the Veterinary School of the University of Pennsylvania, gave a talk on the "Pathology of the Reproductive Organs of Farm Animals." Dr. Underhill brought out many points of interest pertaining to this subject, which were thoroughly discussed by the members present.

A very interesting talk was given by Dr. H. R. Church, Deputy State Veterinarian, who discussed "The Eradication of Tuberculosis in Cattle." He also talked on the quarantine against rabies in the Wyoming Valley.

The next meeting, to be held June 25, 1927, at Montrose, will be in the form of a summer outing, with the wives and friends of the veterinarians present.

THOS. D. JAMES, *Secretary.*

KEYSTONE VETERINARY MEDICAL ASSOCIATION

The April meeting of the Keystone Veterinary Medical Association, held in Philadelphia, Pa., was devoted to poultry diseases. On the program were Dr. E. L. Stubbs, director of the Pennsylvania Bureau of Animal Industry Laboratories; Dr. A. D. Goldhaft, a practitioner of Vineland, N. J., who has an extensive poultry practice; Dr. James J. Black, in charge of poultry pathology at the Vineland Training School, Vineland, N. J., Dr. R. O. Biltz and Dr. H. W. Herriott, of the Pennsylvania Bureau of Animal Industry Laboratories. Drs. Biltz and Herriott gave demonstrations of the methods of testing poultry for tuberculosis and bacillary white diarrhea.

SOUTHEASTERN MICHIGAN VETERINARY MEDICAL ASSOCIATION

The regular monthly meeting of the Southeastern Michigan Veterinary Medical Association was held at the Department of Health headquarters, Detroit, May 11, 1927, with twenty-seven members in attendance. The usual dinner preceded the business session and program.

The film "Clean Herds and Hearts," secured from the United States Department of Agriculture, was shown. Dr. Z. Veldhuis was elected to membership.

Dr. Frank W. Schofield, of the Ontario Veterinary College, Guelph, addressed the meeting on two rather obscure diseases, which he had investigated at different times. The first one, believed to be the condition described in text-books as malignant

catarrhal fever of cattle, resulted in a rather heavy loss in one locality in the province of Ontario. The disease was characterized by high temperature, mucous membranes highly injected, muscular tremors, congestion of the vaginal mucous membrane, opacity of the cornea, followed by blindness, fibrinous exudate in the anterior chamber of the eye and the formation of diphtheritic membranes. Several of the Detroit veterinarians reported having encountered cases of the same disease in southeastern Michigan on different occasions during the past twenty years.

The other disease described by Dr. Schofield was called "horse cholera" or "horse typhoid." The disease has made its appearance on a number of occasions, usually during the months of June and July, in Kent and Essex counties, between Amherstburg and Harrow, in the province of Ontario. Circumstantial evidence tends to incriminate two possible causes—the June fly and new hay. In the locality mentioned, owners of horses refer to the dry and wet types of the disease, which is characterized by the following symptoms: depression, anorexia, congestion of the eyes, increased peristalsis, accompanied by purgation but not by colicky pains. The death-rate varies. Postmortem lesions include marked inflammation of the intestines and peritoneum, cloudy swelling of the parenchymatous organs, marked degeneration of the heart, almost to the point of necrosis in some cases. The lesions are those of an acute infection. Dr. Schofield said that it had been his observation that the incidence of the disease increased as it approached the shore of Lake Erie. Although many cases appeared on farms in the country, very few cases were found among horses stabled in the cities.

Dr. Schofield then showed some lantern-slides of conditions in Japan and Korea, countries which he visited recently. These proved to be very interesting and Dr. Schofield's characteristic way of relating his experiences proved to be very enjoyable to his audience.

H. Preston Hoskins, *Secretary.*

JOINT MICHIGAN MEETING

The 1927 Lake States Tuberculosis Eradication Conference will be held in conjunction with the annual meeting of the Michigan State Veterinary Medical Association, June 27-28-29, at Lansing and East Lansing, Mich.

NECROLOGY

HENRY EDWARD HOLDEN

Dr. H. E. Holden, of Springfield, Mass., died April 3, 1926. He was a graduate of the American Veterinary College, class of 1890 and joined the A. V. M. A. in 1892. He was a member of the Massachusetts Veterinary Association.

CHARLES C. CATTANACH

Dr. Charles C. Cattanach died at West End, N. J., April 26, 1926, aged 68 years. He was a graduate of the American Veterinary College, class of 1879 and had practiced continuously in New Jersey for almost half a century. He was official veterinarian of the Monmouth County (N. J.) Horse and Dog Show for the past thirty years.

WILLIAM H. NICE

Dr. William H. Nice, for many years in general practice in Germantown, Philadelphia, died June 30, 1926. He was a graduate of the American Veterinary College, class of 1887.

J. J. IRWIN

Dr. J. J. Irwin, of Teulon, Manitoba, died in the General Hospital, Winnipeg, May 1, 1927. He was a graduate of the Ontario Veterinary College, class of 1884, and had practiced in the Stonewall and Teulon districts for over twenty-five years.

ROBERT H. APITZ

Dr. Robert H. Apitz, of Hancock, Minn., died suddenly at his home, March 3, 1927. He was a graduate of the Kansas City Veterinary College, class of 1910, and conducted a very successful general practice at Hancock. He is survived by his widow and three children.

THOMAS H. HOPE

Dr. Thomas H. Hope, of Waukegan, Ill., died at the home of his son, in Chicago, April 22, 1927. Dr. Hope was the sole surviving member of the 1869 graduating class of the Ontario Veterinary College.

Born at Ayr, Ontario, January 31, 1846, he returned there to practice following his graduation. In 1884 he removed to Waukegan, Ill., and was in active practice until a few years ago, when he retired.

Dr.Hope is survived by his widow and one son.

LYMAN E. McDONALD

Dr. Lyman E. McDonald, of Monroe, La., lost his life, May 14, 1927, as the result of the flood conditions in the lower Mississippi Valley. He was drowned in the Ouachita River, four miles south of Monroe. He was on his way to New Orleans, in a skiff, which caught fire. Dr. McDonald made a brave effort to save the burning boat but was unsuccessful. He jumped into the river and lost his life in the swollen waters.

Born at Jonesboro, La., March 21, 1892, Dr. McDonald was graduated from the Kansas City Veterinary College, in 1914. He entered the service of the Bureau of Animal Industry and was engaged in tuberculosis eradication work when he resigned, several years ago, to engage in dairying in Ouachita Parish, Louisiana, a few miles south of Monroe.

Dr. McDonald joined the A. V. M. A. in 1919.

MARRIAGE

Helen Janet Adams, daughter of the late Dr. John W. Adams, to William Ross Thompson, of Warsaw, N. Y., at Swarthmore, Pa., April 16, 1927. They will reside at Warsaw, N. Y.

BIRTH

To Dr. and Mrs. D. W. Ashcraft, of Columbus, Ohio, twins, Derwin Willoughby, Jr., and Merwin Gorrell, May 9, 1927.

PERSONALS

Dr. Gerald P. Finck (U. P. '25), of Hatboro, Pa., is studying human medicine.

Dr. H. F. Wilder (Corn. '26) is assisting Dr. F. L. Stein, of Rochester, N. Y.

Dr. DuBois Jenkins (Corn. '18) has removed from Cobleskill to Oxford, N. Y.

Dr. Earl F. Hoover (K. S. A. C. '24) is assisting Dr. B. F. Pfister, of Kansas City, Mo.

Dr. H. W. Graves (A. P. I. '23) owns and operates the City Abattoir of Union, S. C.

Dr. Milton R. Fisher (O. S. U. '25) is with the City Health Department of Paducah, Ky.

Dr. G. L. Kreiger (K. S. A. C. '24) is with the DeVine Laboratories, at Goshen, N. Y.

Dr. A. M. Mills (Corn. '20), formerly at Oxford, N. Y., is now at Washingtonville, N. Y.

Dr. Rease Mitcham (St. Jos. '23) has removed from El Dorado, Ark., to Seminole, Okla.

Dr. Bruce Kester (O. S. U. '25) is engaged in municipal food inspection work in Dayton, Ohio.

Dr. D. C. Oakleaf (Colo. '25) is studying human medicine at the University of Colorado, Denver.

Dr. Florian J. Olbrich (Iowa '22) is a sophomore in the Jefferson Medical College, Philadelphia.

Dr. B. E. Grover (Chi. '12) has removed from West Branch, Iowa, to Pleasantville, same state.

Dr. Robert O. Hughes (Iowa '23) is studying human medicine at the University of Iowa, Iowa City.

Dr. L. W. Goodman (Corn. '26) is now located at 45 Cutter Mill Road, Great Neck, Long Island, N. Y.

Dr. Hazen S. Atkins (Mich. '23) is chief of the Dairy and Food Division, Department of Health, Pontiac, Mich.

Dr. S. C. Cheng (Iowa '26) is taking a rest at Trudeau, N. Y., preparatory to taking postgraduate work next year.

Dr. Edward Everett (A. P. I. '22) is Associate State Veterinarian of North Carolina, with headquarters at Raleigh.

Dr. John H. Gillmann (St. Jos. '17), formerly located in Memphis, Tenn., now receives his mail at Jackson, Tenn.

Dr. T. A. Reagan (A. P. I. '22) has been athletic director at the Gadsden (Ala.) High School for the past three years.

Dr. Harvey L. Fell (U. P. '25), of Wilmington, Del., conducts a boarding and riding stable in connection with his practice.

Dr. John R. Harney (Mich. '24) is with the New Jersey Bureau of Animal Industry, with headquarters at Bridgeton, N. J.

Dr. G. S. Harshfield (O. S. U. '26) is engaged in tuberculosis eradication work in Ohio, with headquarters at East Liberty.

Dr. R. R. Clark (K. C. V. C. '05), formerly of Williamsburg, Va., gives a new address: 1329 24th Street, Newport News, Va.

Dr. Arthur Delez (Colo. '25) is taking work for the degree of Master of Science, at the Michigan State College, East Lansing.

Dr. James J. Bogan (Corn. '23) is located at Catskill, N. Y., as County Veterinarian, engaged in tuberculosis eradication work.

Dr. J. S. Barber (Chi. '10), formerly of Princeton, Iowa, has opened a small animal hospital at 14 Washington St., Central Falls, R. I.

Dr. W. A. Aitken (Iowa '17) has been made Professor of Veterinary Anatomy, Division of Veterinary Medicine, Iowa State College.

Dr. Earl B. Leneker (O. S. U. '22), of Cherry Valley, N. Y., writes that conditions in general practice are good and steadily getting better.

Dr. W. L. Parrott (K. S. A. C. '26) is with the New Jersey Bureau of Animal Industry, engaged in field work, with headquarters at Trenton.

Dr. Thomas S. Carlisle (U. P. '01), of Chestnut Hill, Pa., has completely recovered from his recent illness and is now back in active practice.

Dr. Lyle Pratt (U. P. '18) has located at Pottstown, Pa., for general practice. He was formerly with Dr. I. V. Stoll (U. P. '16), of Rome, Pa.

Dr. C. R. Peterson (Mich. '24), who is in general practice at Chatsworth, Ill., writes that practice is good and that he has no complaints to offer.

Dr. A. B. Davis (Ga. '22), of Donaldsonville, Ga., is interested in the organization of a system of municipal sanitary control work for his city.

Dr. C. A. Brandly (K. S. A. C. '23), who has been in the employ of the New Jersey Department of Agriculture, is now located at Weehawken, N. J.

Dr. V. W. Knowles (K. C. V. C. '07), who has been practicing in Miami, Florida, for some time, gives a new address: 1958 Thomas Street, Hollywood, Fla.

Dr. J. R. Roche (Iowa '23) has resigned from the U. S. Bureau of Animal Industry, to accept a position with the Sioux City Serum Company, Sioux City, Iowa.

Dr. G. R. Penhall (Ont. '22) is engaged in field work for the Health of Animals Branch, Canadian Department of Agriculture, with headquarters at Toronto, Ont.

Dr. William R. Crawford (Corn. '24) is with the Maryland State Department of Agriculture, engaged in tuberculosis eradication work, with headquarters at Westminster, Md.

Dr. R. G. Chapman (Ont. '26) is with the Health of Animals Branch, Canadian Department of Agriculture, engaged in meat inspection, with headquarters in Toronto, Ont.

Dr. B. A. Bescoby (Ont. '08), who has been living in France for a period of two years, has returned to the province of Manitoba and located at Emerson for the practice of his profession.

Dr. Lester H. Bennett (O. S. U. '23) has full charge of 458 head of live stock at the Louisiana Training Institute, at Monroe, La., besides conducting his general practice at that place.

Dr. J. V. Duckworth (A. P. I. '23) is in practice at Hazelhurst, Miss. He was with the State Live Stock Sanitary Board of North Carolina for nearly two years, prior to resuming practice.

Dr. E. P. Johnson (Mich. '25) is pathologist for the Michigan Department of Agriculture, with headquarters in the Department of Bacteriology at Michigan State College, East Lansing.

Dr. R. M. Shreve (Chi. '10), of Panora, Iowa, was called away from his practice during April on account of the serious illness and death of his mother. Our sympathy is extended to Dr. Shreve.

Hon. D. H. McFadyean (Ont. '80), honorary member of the Manitoba Veterinary Association, has received the Conservative nomination to represent Emerson in the elections this month.

Dr. E. J. Jelden (K. S. A. C. '22) is pursuing a course in human medicine at the University of Kansas, Lawrence. During vacations he practices as an assistant to Dr. A. H. Gish, of El Dorado, Kan.

Dr. C. E. Reckin (Ont. '26) is with the Canadian Milk Products, as veterinary inspector, on health inspection of cattle and dairy sanitation work at all plants, with headquarters at Tillsonburg, Ont.

Dr. F. Ashton Clarke (A. P. I. '24) supervises the work of the municipal milk inspectors of Montgomery, Ala., which city has in force the standard milk ordinance of the U. S. Public Health Service.

Dr. John V. Nevitt (Corn. '24) is Dairy Development Agent for the Missouri Pacific System lines. His headquarters are in Carthage, Mo., and his territory embraces Oklahoma, Missouri, Kansas, Arkansas and Louisiana.

Dr. W. S. Plaskett (McG. '93), of Clinton, Mass., accompanied by Mrs. Plaskett, is on a vacation trip planned to extend over four months. They will include California in their itinerary, returning through Canada.

Dr. John P. Hutton (O. S. U. '11), of Michigan State College, has been elected permanent secretary of the Michigan State College R. O. T. C. Horse Show. The fifth annual exhibition was held at East Lansing, May 30-31, 1927.

Dr. J. A. Munn (Ont. '08), of Carman, Manitoba, has received the Progressive nomination for the constituency of Dufferin in the elections to be held this month, as a supporter of the Bracken Government in the local legislature.

Dr. J. G. Hardenbergh (U. P. '16), who has been at the Mayo Foundation, Rochester, Minn., for a number of years, has resigned to accept a position with the Walker-Gordon Laboratory Company, Plainsboro, N. J., June 1.

Dr. R. G. Flowers (Ont. '06) acted as judge and veterinarian at the third annual Fort Worth (Texas) Pet Stock Show, held at the Fort Worth Coliseum, May 5-6, 1927. The Show was sponsored by the Recreation Department of the city.

Dr. Harold M. DeVolt (Corn. '23), who has been at the State Institute of Applied Agriculture, Farmingdale, Long Island, N. Y., has accepted a position in the Department of Bacteriology and Sanitation, University of Maryland, College Park.

Dr. A. A. McMurray (O. S. U. '24), formerly with the New Jersey Bureau of Animal Industry, has accepted a position with the Supplee-Wills-Jones Milk Company, of Philadelphia, as field veterinarian, with headquarters at Mercersburg, Pa.

Dr. A. E. Drexel (Ga. '24) is studying medicine in the Medical Department of the University of Georgia, at Augusta. He spent eight months in the Bureau of Animal Industry, on meat inspection work, before taking up the study of medicine.

Dr. T. E. LeClaire (Laval '90), of Calgary, Alberta, who has been spending the winter in Mexico, Arizona and California, left Los Angeles recently for New Orleans. From there he will sail for New York and Montreal and will either motor or travel by rail and water on his return to Calgary. He expects to be home by July 15, 1927.